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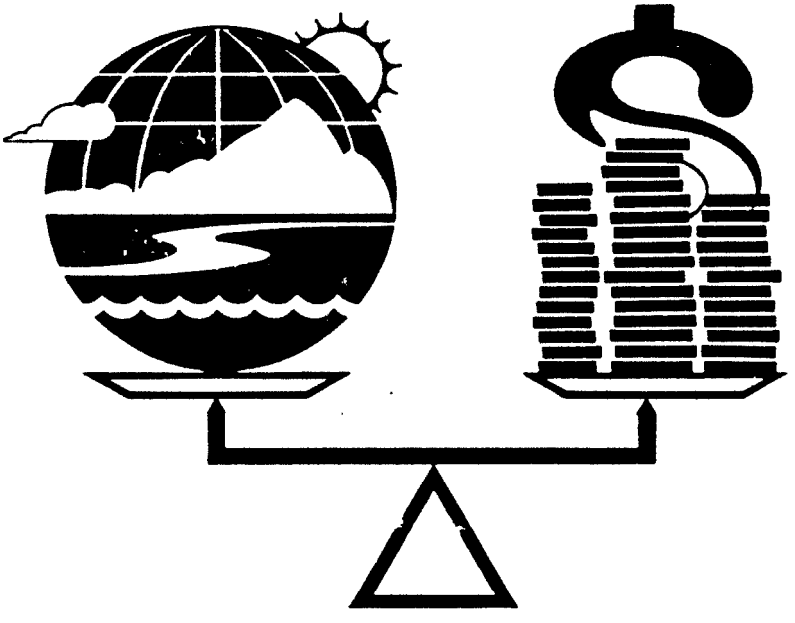

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AERE Workshop

Estimating and Valuing Morbidity in a Policy Context

Proceedings

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THE ROLE OR EPIDEMIOLOGY IN DEVELOPING USEFUL DATA FOR PUBLIC HEALTH POLICY

by

Daniel A Hoffman

The Role of Epidemiology in Developing Useful Data for Public Health Policy

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INTRODUCTION

In the last two decades, the role of epidemiology in providing data for public health policy makers has become more prominent than any time in the history of this discipline. Its greatest advantage is that it provides direct human evidence of the health outcomes from various environmental exposurs, unlike animal models. However, there are many caveats that need to be attached to these data. The objective of this paper is to review some of the basic limitations to the epidemiologic method, both in study design and in interpretation of the data. The perspective that I will present is that of an epidemiologist in a public health agency. The Centers for Disease Control does not engage in the development of regulations or have a large program in risk assessment.

Our principle function is to serve the state and local health departments by offering advice and assistance when necessary through field investigations of potential public health problems-in our case, environmentally-induced disease. This involves the identification of study hypotheses, designing the study, developing the necessary survey instruments, collecting health data, analyzing this data, and finally offering our interpretation and recommendations. Basically we engage in the classical epidemiologic method of hypothesis generation and testing through field investigations. Consequently, my talk today is focused on the techniques involved in acquiring these data, and examining the strengths and weaknesses of these data as they relate to various interpretations of their meaning for use in risk assessments, regulatory actions, or public health policy decisions.

DESCRIPTION OF EPIDEMIOLOGIC METHODS

The assessment of effects on humans of various environmental exposures relies heavily upon the results from testing of animal models and clinical and epidemiological studies. However, the most important advantage that epidemiological studies have over animal investigations is that they provide direct evidence of the effects of toxic exposures in humans. Conversely,

human studies are difficult to conduct properly and the interpretation of the results from these studies makes life difficult for both regulators and policy makers. Part of the problem in interpretation stems from the design of these studies which can be very complex. Another problem is dealing with the inherent biases which inevitably creep into the interpretation of the data, no matter how thoroughly these have been addressed either in the study design or analysis. This stems from the fact that, with the execption of clinical trials, epidemiological studies are observational by nature, not experimental. Not only do humans vary widely in their response to toxic agents but they vary also in their capacity for response as well as in their exposure to factors such as alcohol and tobacco, which may may greatly modify the nature or severity of their responses to toxic exposures. One example would by the relation between radon exposure and cigarette smoking which could either be additive, submultiplicative or mulplicative depending upon which data is reviewed and which model is applied to that data.

Despite these difficulties, techniques for the evaluation of data from human studies have been developed and refined. The epidemiological method has matured to the point that it has withstood the criticism that it is

incapable of establishing the etiology of disease. Epidemiological inferences have been sustained and corroborated by the results of toxicological and biochemical studies, and epidemiology has proven to be a powerful tool for the exploration of both qualitative and quantitative cause-and-effect relationships between environmental exposures and human disease. However, there is still much to be done, especially at the rather low levels of exposures that most human populations experience, to further refine the tools of epidemiology. I would now like to briefly discuss some of the various study designs used in epidemiology. Next I will address some of the sources of bias in epidemiologic data, and conclude with a discussion on interpretations of causality based on data derived from epidemiological studies. Two areas of study which I will not discuss in any detail today are the appropriateness of animals models as they apply to risks in humans and the use of biomarkers as indicators of risk in epidemiologic studies. The majority of our experience at CDC has been concerned with collection and interpretation of epidemiologic data so the principal focus of my talk will be on that process.

The most commonly used designs in epidemiology are: 1) case reports; 2) ecological or correlational studies; 3)

cross-sectional studies; 4) case-control studies; and 5) cohort studies.

1. CASE REPORTS

Case reports identify one or more cases of a disease that have been detected by clinicians, by company or union officials, or by through active surveillance or passive reporting such as cancer registries. The first recorded case studies of environmental disease were Sir Percival Pott’s observations of scrotal cancer among chimney sweeps in London. Publication of such case reports often constitutes the first recognition that a problem of environmnetally induced disease exists, and subsequent epidemiological assessment proceeds from this recognition. A more recent example includes the first recorded cases of AIDS by clinicians at UCLA medical center in 1978. In a case series, an inference of causal association between causation and an environmental agent is based on the plausibility of the following considerations: clustering of the cases in a limited time frame; the relative rarity of the types of diseases observed; a history of common environmental exposure; an the apparent strength of the association. The most common use of case reports are hypothesis generation, surveillance, and case registries

Surveillance

The case report has historically been an important" surveillance tool, especially for recognition of infectious diseases. Occupational case reporting has been useful in terms of reporting occupational injuries for workman's compensation, but not so much for occupational diseases due to the long latency period between exposures and disease. A more recent use of case reports as a surveillance tool is for the identification of senital health events. These are cases of disease associated with well-characterized causes whose appearance signals a breakdown in mechanisms for disease prevention. This method has been applied with success in the reduction of maternal and infant mortality and has been extended to such environmental illnesses as lead poisoning.

Case Registries

Other surveillance systems relying on case reports include case registries, such as the CDC Dioxin Registry or workers suspected of having been exposed to dioxin. These exposure registries perform the task of grouping potentially high-risk populations for future epidemiological studies.

An advantage of case reports over most other types of epidemiological studies is their low cost. In addition, a

short lag time between identification of cases and dissemination of information is more typical of case reports. However, relying on case reports as an early warning system is less useful when:

1) the cases are sporadic;
2) the relative risk is low;
3) the outcome is a common disease or a symptom with multiple common etiologies such as lung cancer or heart disease;
4) there is a long latent period between exposure and effect; and
5) there is a continuum of disease and health and no clear distinction between cases and noncases is possible, for example, premalignant dysplasia and carcinoma in situ.

In addition, case reports can provide only a rough estimate of disease frequency, in that they give no information on the size of the population at risk and thus make it impossible to calculate a disease rate. Finally, case reports are difficult to generalize to a population since the population from which the cases are identified is not usually well defined.

2. CORRELATIONAL OR ECOLOGIC STUDIES

Another type of descriptive tool used by epidemiologists is the so-called correlational or ecologic study, which

uses data from entire populations to compare disease frequencies between different groups during the same period of time or in the same population at different points in time.

As an example of the former, correlational studies have suggested that various dietary components, in this case per capita meat consumption, may be risk factors for colon cancer. Figure 1 shows the correlation between per capita consumption of meat and rates of colon cancer in women from a large number of countries. As apparent from this figure, the rates of colon cancer are lowest in countries with the lowest per capita meat intake and vice versa.

Figure 2 illustrates the change in disease frequency within the same population over time. In this slide, the difference between tge approximately 820,000 deaths from coronary heart disease that would have been expected in the United States if the 1968 rates had continued to apply and the approximately 630,000 deayhs actually observed. Such data suggest two possible explanations: 1) that the decline in deaths from coronary heart disease could be due to prevention due to improvements in life-style habits and consequent risk factor reduction, and 2) that while the rates of CHD did not decline, persons were surviving longer due to improvements in medical management of CHD.

While correlational studies are useful in developing hypotheses for study, they cannot be used to test them because of a number of imitations inherent in their design.

1) Correlational studies refer to populations rather than to individuals. Therefore, it is not possible to link an exposure to occurrence of disease in the same person.

2) The distribution of other risk factor's which may account for different rates of a disease, may be differentially distributed among populations. This is known as the "ecologic fallacy".

3. CROSS-SECTIONAL STUDIES

Another type of descriptive study design is the cross-sectional survey, in which the status of an individual with respect to the presence or absence of both exposure and disease is assessed at the same point in time. For example, the Health Interview Survey is a national cross-sectional study that periodically collects extensive information by questionnaire from a sample of over 100,000 persons throughout the United States. These studies often rely on personal interviews or

questionnaires to obtain demographic information, symptomatic, and exposure data on clinical evaluations based on physical examinations and laboratory and environmental sampling data to identify the characteristics of the sample population and to quantitate exposure to potential risk factors. An advantage of the cross-sectional survey is the rapid estimation of numerator values for determining frequency or prevalence rates of both exposure and effects. Limitations of this method include the inability to distinguish whether the exposure preceeded the development of disease or whether the presence of disease affected the individual's level of exposure, since exposure and disease are assessed at the same point in time. Cross-sectional approaches have limited usefulness in cancer studies because of the usual low prevalence of cases. It is also extremely difficult to quantify exposure in cross-sectional studies. However, for factors that remain unaltered over time, such as sex, race or blood group, the cross-sectional survey can provide evidence of valid associations.

Five common pitfalls can be found in the cross-sectional method. These are:

1) Selection bias, in that a nonrepresentative sample of the population may be surveyed, limiting the generalizability of the survey results;

2) Confounding bias, which can result for factors related to both exposure and outcome, such as age;

3) Inadequate sensitivity of the survey instruments. This includes specificity, which is the ability to detect "true" negatives, and sensitivity or the ability to detect "true" positives;

4) Lack of standardization of the instruments used for data collection, which may prohibit the pooling of data from multiple surveys; and

5) Inadequate validation of either exposures of health outcomes, resulting in misclassification of either category.

Summarizing, in general, cross-sectional studies are useful for raising the question of the presence of an association rather than testing a hypothesis.

The next two types of epidemiologic studies are observational in design. These are the case-control study and the cohort study.

In theory, it is possible to test a hypothesis using either design strategy. In practice, however, each design offers certain unique advantages and disadvantages. In general, the decision to use a particular design is based

on the features of the exposure and disease, the current state of knowledge, and logistic considerations such as available time and resources.

4. CASE-CONTROL STUDIES

In the case-control study, a case group or series of patients who have a disease of interest and a control or comparison group of individuals without the disease are selected for investigation, and the proportions with the exposure of interest in each group are compared. Lung cancer patients, for example, can be compared to persons without that disease for differences in exposures, such as cigarette smoking, occupational exposures, and radon levels in the home.The relative frequency of distribution of the exposure in the case and control groups is usually evaluated by computing an odds ratio which is defined as the product of the number of exposed cases and unexposed controls divided by the product of the unexposed cases and exposed controls. This is also somtimes known as the cross-product odds ratio because of the manner in which it is calculated.

Case-control studies can be conducted relatively rapidly. Many simultaneous exposures can be evaluated in relation to even the rarest disease. Howver the sequence of

exposure-health event is often difficult to assess if the case population includes patients selected from historical records. If the disease studied is rapidly fatal, interviews with surrogate respondents may be required which may result in misclassification of exposures. The individual exposure status is often difficult to quantify with any precision, especially in environmental studies, and control of possible confounders may require a complex design or analysis. Consequently, only environmental exposures with a high prevalence and relative strong toxic effect are effectively studied by the case-control method.

5. COHORT STUDIES

In a cohort or follow-up study, the study population is divided on the basis of exposure status. For example, in a recent study of the health effects of volatile organic compounds in Michigan, we assembled study cohorts on the basis of whether or not VOC'S were detected in their well water and if they had lived for a specified period of time in the study area. Residents who had moved away prior to the initiation of the study were still eligible for inclusion in either the exposed or unexposed cohorts. Once the exposure status of the study cohorts has been determined, which is sometimes quite complex and can

result in misclassification of exposure status thus biasing the study outcome towards the null hypothesis of finding no effect, the history of disease is determined in both the exposed and unexposed groups. The rate of disease in the exposed group is compared to that in the unexposed group resulting in a relative risk of disease which could be due to the exposure being studied. This is also called the rate ratio since it is simply the ratio of two incidence rates. Both of these measures of association include a factor for follow-up time known as the person-year. This is simply defined as the interval from the time exposure began to the date of diagnosis of disease, death, loss-to-follow-up or, if disease-free, an arbitrary date.

The strengths of the cohort approach include the following:

1) the sequence of exposure and health outcome can be studied;

) many health outcomes can be evaluated with regard to the one exposure of interest (although this may have become a problem in some studies as multiple comparisons inevitably lead to at least one “significant” finding);

3) the initial exposures can be quantified through historical records or even more so if there is a

biologic marker of exposure such as blood or bone lead levels;

4) rare exposures can be studied;

5) collection and analysis of potential confounding factor is possible; and

6) absolute risks may be calculated for use in public health prevention strategies.

Some of the drawbacks to the cohort approach are the expense and difficult logistics of these studies, the potential for misclassification of exposure and disease outcome resulting in a biased estinate of risk, and the inability to study rare disease because of the very large populations necessary for study. This latter drawback is. important in studying the effects of low-level environmental exposures. Because the anticipated risk of these exposures is low, very large numbers of exposed persons are required for study if the outcome is to have any decent statistical power.

PROBLEMS IN CURRENT STUDY DESIGNS

From the previous discussions, four areas of major problems become evident: 1) the assessment of the exposure-response sequence; 2) quantification of exposure; 3) recognition of bias and confounding; and 4)

quality and validity of data. Clearly, a very complex study design may be required to yield useful results.

Measures to improve the usefulness of human studies for risk assessment purposes include the extension of the duration of follow-up time, assessing the time component in exposure and disease diagnosis, focusing on potentially high-risk populations for study, and quality assurance of information on exposure and disease. While most of these measures are in the area of logistics and funding, an important exception is improvement of the quality of the exposure data.

In the past decade, development of environmental exposure mesures has been very rapid. Detection limits for chemicals in environmental media have dropped by three to four orders of magnitude, and the progress of tests for some chemicals in biological media is almost as impressive. The detection limits for dioxin in sera, for example, is now measured in parts per quadrillion. Unfortunately, little progress to date has been found to be of practical use in epidemiologic analysis and risk estimation. For instance, issues of background levels, biological persistence, adaption mechanisms, absorption kinectics, saturation of metabolic pathways, and the impact of an individual’s characteristics on the pathogenetic process have not been addressed in most

epidemiologic study designs, and, for the most part, have yet to enter the area of regulatory risk assessment.

There are other practical exposure issues that need to be addressed such as noncontinuous or fluctuating exposures, the cause of interspecies differences, and whether or not an observed dose-response relationhip is stable over a wide range of dose levels. We will also see an increasing demand to incorporate quality assurance and quality control in epidemiologic studies with regard to matters other than laboratory work. For example, it is of utmost importance to make certain that the disease of concern is following and not pre-dating exposure. Finally, there is the issue of the quality of the diagnostic criteria for a case or a non-case.

The quality of diagnosis becomes a very central issue when it comes to scenarios of localized environmental pollution, for example, at a chemical dump site, and residents with nonverifiable and subjective complaints, which may be real to them, sucah as headache, fatigue, nausea, chest pain, and loss of libido. Currently, there is an inclination among epidemiologists to ignore or disqualify this so-called “dump-site syndrome” from serious study. However, such an attitude is usually followed by a deterioration of a conflict situation between citizens and authorities. There are many

instances where eventually epidemiologists have been forced by heavy and relentless public and political pressures to conduct studies of such perceived illnesses. In doing so, they will have to derive methods to cope with non-verifiable health outcomes, while maintaining scientific integrity and credibility. In theory, it should be possible to either solve the problems with statistical tools, or by developing tests for the kinds of complaints often described as emotional or behavioral. CDC staff are currently developing and applying such tools to several large studies.

Statistical methods usually fail since the situation at a dump site is inherently associated with an abundance of negative publicity, usually in the direction of stating the association of voiced complaints with exposure, or even just living near a dump site, as a fact. This scenario often results in serious reponse biases for persons who perceive they may be exposed. I do not forsee that behavioral toxicology, an exciting new field of research, can provide us in the near future with the appropriate scientific tools to address currently nonconfirmable complaints.

DEVELOPMENT OF MOLECULAR EPIDEMIOLOGY

A special problem, both in animal and human studies, is

that current designs deal with observed disease, which is a more or less advanced stage of a toxic effect. In animal studies, most diseases are observed in moribund or sacrificed animals. In humans, disease detection is usually in an earlier phase by virtue of man’s ability for detailed communication. However, even common diseases such as cancer, arthritis, hypertension, and diabetes still pose unresolved problems in assessing the date of onset. Estimates of this date may differ by many years, and this would offset greater accuracy in exposurement. The logical response to this problem is to develop techniques to diagnose the disease in the earliest possible stage. But the question then arises: “What is earliest possible?" An aggressive biopsy regimen for diseases such as cancer and kidney disease may shift the date of diagnosis from months to years earlier. Certain inborn metabolic disorders can now be detected prenatally. The use of electron microscopy has brought us closer to the early onset of renal disease. Unfortunately, these striking improvements in early diagnosis require invasive procedures. This is a serious handicap to epidemiologic studies, especially those involving environmental rather than clinical or occupational exposures. This explains the increasing interest of epidemiologists and risk assessors in the use of biomarkers indicating past exposures or early stages of tissue dysfunction, for example, DNA-adducts.

VALADIATION OF ASSUMPTIONS FROM EPIDEMIOLOGIC STUDIES FOR REGULATORY PURPOSES AND PUBLIC HEALTH POLICY DECISIONS

Finally, I would like to discuss the interest of epidemiologists in the validation of a number of assumptions used in risk assessments for regulatory purposes or public health policy decisions. One of these is the assumption that the presence of a toxic chemical in the environment automatically implies exposure, and that that body dose is proportional to environmental concentrations. This assumption leads to the often-used, but nevertheless incorrect practice of assuming that the concentration of a chemical in media such as soil, air or water is a direct measure of the amount of chemicals absorbed in the human body. Worse, without much thought it is often considered identical to the challange to the organ or tissue interest when determining acceptable exposure levels. Studies into the relation between environmental presence, human exposure, and organ-specific dose are increasing in number.
The findings from these studies have sometimes been contrary to expectation. For example, at the CDC, studies have shown that the concentration of arsenic, PCB’S, mercury, and lead in the soil of a neighborhood is only partly related if at all to the levels in the biologic specimens of residents. In this light, it is important to recognize the importance of well-conducted research with

negative findings. Such research is critical to our understanding of the effects of toxicants on human biology. Moreover, such findings help concerned scientists to inform the public of true risks and allay undue anxiety. Indeed, despite the abundance of available data to date, the relation between environmental concentrations of chlorinated hydrocarbons such as DDT, dioxin, and PCB'S, and human sera or adipose samples, remains unclear, and the relation of these levels of body burdens to clinical disease remains uncertain.

To date, epidemiologic studies almost never prove cause and effect, though in a few instances, reasonable people would accept some of them as such. For example, in looking at the pathway of exposure and body burden, the association of the reduction of lead used in gasoline production and the reduction of mean blood lead levels in the U. S. population is striking. Over a 4-year period when the lead phasedown in gasoline was occurring, we were conducting a study of blood lead levels in the U. S. population using data from the Second National Health and Nutrition Examination Survey or NHANES-2, an example of a cross-sectional study. Two things, declining blood lead levels and lead used in gasoline production were highly correlated. We removed over 100 potentially confounding variables from this association in the analysis and the coefficient of correlation did not appreciably change. Yet many epidemiologists stated that this did not provide

adequate evidence of cause and effect. The only way to unequivocally prove cause and effect in this situation would be to conduct an experimental study where children were placed in chambers and breathed air with different lead levels and then measure their blood lead levels. This experiment, of course, would be entirely unethical and would not be supported by society. Studies conducted in humans must use only inadvertent exposure or natural experiment s“ such as that occurred with water fluoridation and dental carries.

Proper use of epidemiologic data can lead to important collective public health benefits. On the other hand, to press such data into service to respond to causal effects for an individual’s disease holds high potential for misuse of the data.

We will continue to respond to specific incidents of human exposure to toxic or hazardous substances. We will also continue our efforts, through epidemiologic techniques, to measure both the immediate and long-term health effects and to make sound recommendations for the attenuation of these potential risks.

Although the results of such epidemiologic investigations may not provide the conclusive answers about health risks from environmental exposures, which are now in such

demand and so prevalent in the media, we have hope that we can study and detect these associations where they exist, so that prudent public health actions can be taken. Thus, we see the ultimate role of epidemiology as one of prevention, which is the most effective public health policy to implement.

SUGGESTED READINGS

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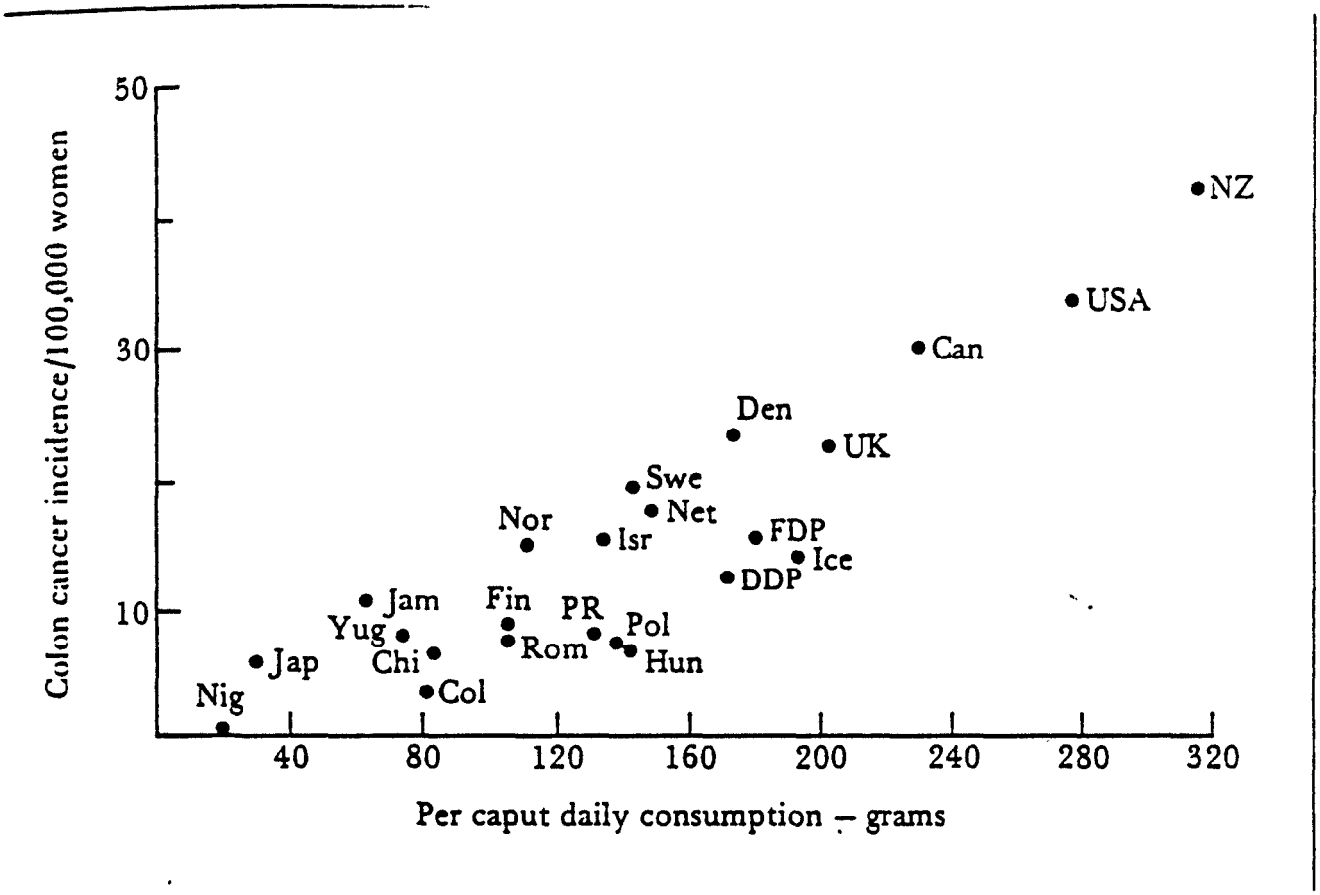

Figure 1. Colon Cancer Incidence Rates/100,000 by Per-Capita Meat Consumption (grams) for selected countries

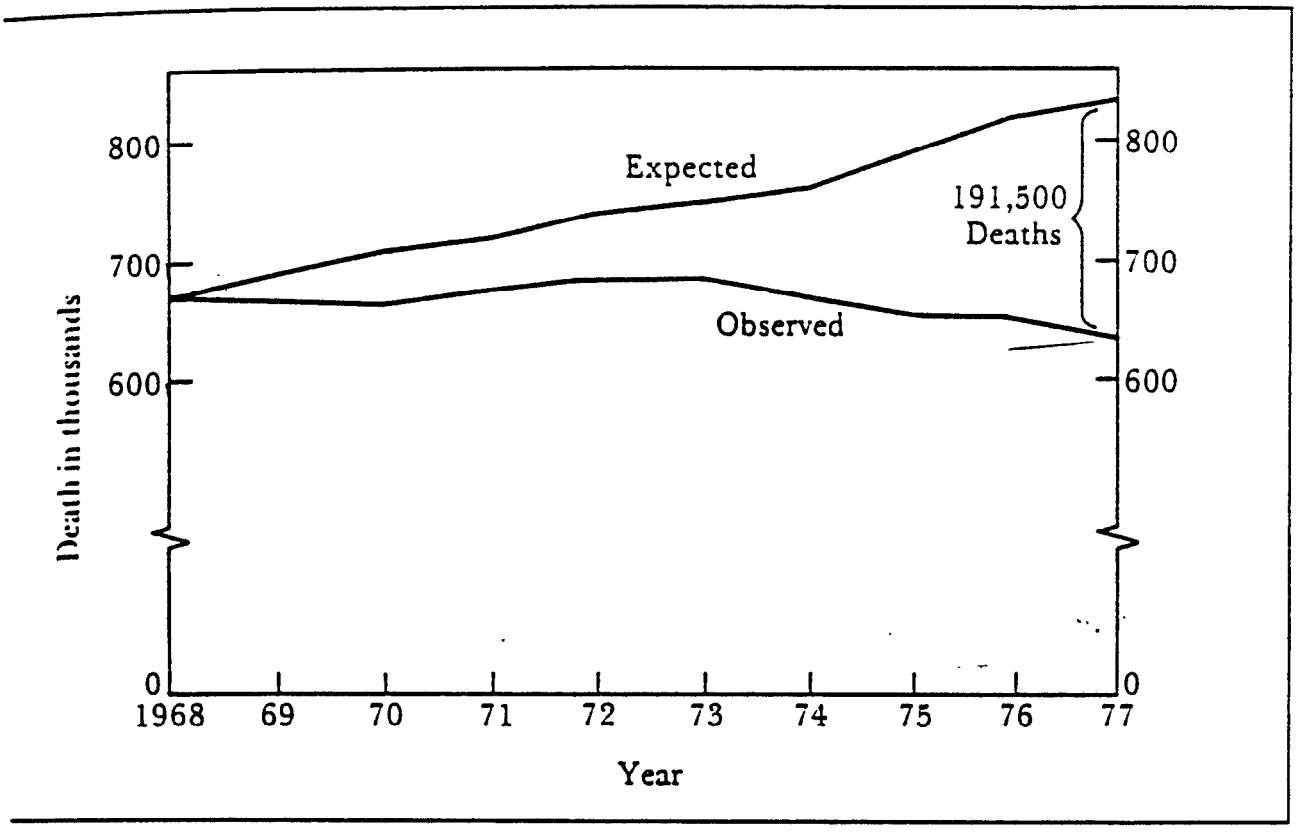

Figure 2. Difference Between Observed and Projected Mortality Cases from Coronary Heart Disease, United States (If deaths in 1977 occurred at the same rates as in 1968)

D R A F T

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Malignant Melanoma Death Rates, Outdoor Recreation and Sun Screens

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May 18, 1989

Disclaimer: This paper reports interim results on research which is still in process. It presents the personal opinions of the author. It has not been reviewed by EPA and does not represent an official EPA position.

Abstract

Previous work has shown that there is a six fold increase in the risk of death from melanoma for white males born in the 1940's when compared to white males born in the 1880’s and 1890’s. For women the same ratio is slightly less than three. Accepting the hypothesis that most melanoma is caused by exposure to solar radiation, an investigation of changes in residence patterns, occupation, and outdoor recreation is made to see if the changes in cohort risk can be explained by changes in factors related to exposure patterns. Household access to automobiles turns out to be the best potential measurable factor explaining outdoor recreation patterns. While no conclusive findings are reached, support is developed for the hypothesis that intense exposure of skin which has not developed natural defenses under low to moderate exposure is the primary risk factor for melanoma. The introduction of sun screens” is associated with reductions in this risk. Since lifetime incidence rates for white males in the 1940’s cohorts will approach 2.5 percent with death rates of about .6 percent, melanoma is a significant public health problem. The paper’s results suggest that a risk communication policy should be aimed at modifying sun exposure habits to reduce intensity of exposure. The association of the automobile with problem exposure behavior suggests a strategy of keeping sun screen in the glove compartment. The payoff from such a policy could be a dramatic reduction in melanoma incidence and death.

INTRODUCTION

Cutaneous malignant melanoma is the most rapidly rising cause of cancer death for white males and the second only to lung cancer for white females.[1] In response to this, there has been much investigation of potential causes.[2] Because melanoma cannot be induced in small laboratory animals by ultraviolet radiation alone, the ability of laboratory research to settle etiologic issues has been sharply limited.[3] Epidemiologic results have been inconsistent, with less melanoma observed on frequently exposed parts of the body, and death rates increasing with latitude in Europe.[4,5] Occupations involving outdoor exposure have been found to be mildly protective.[6] It has been difficult to develop a model which can comfortably explain all of these results. Thus, despite all the work on melanoma to date, there is still a clearly understood feeling by the research community that this is a disease whose etiology is not at all well understood.[8]

The first part of the paper reviews some basic biological and epidemiological results. The next section reports on previous results obtained in this research project. The project has been centered around factors affecting melanoma death rates for US whites between 1950 and 1984. County death rates have been agregated into Standard Metropolitan Areas(1980 definition) and merged with census data on sociodemographic characteristics of the 1980 population, weather data for each city, and model based

estimates of exposure.[9] This data set has been used to investigate the response of death rates to potential exposure, the cohort structure of death rates, and the response of death rates to individual components of the ultraviolet spectrum. For this paper, the data set is used to predict cohort levels of risk, which serves as the basis for the analysis of changes in ecologic risk factors.

The third part of the paper then precedes to examine how factors such as outdoor recreation, outdoor work, and residence have varied over the period for which cohort risk of death of melanoma can be inferred from the data set. First, some measures of how these factors have changed are developed. These are then compared to the summary risk measures for each cohort. Out of this there emerges a fairly clear picture of the kinds of exposure factors that can be related to the observed change in risk. These factors can explain the rise and stabilization of the cohort risk factors. They cannot explain the downturn in risk seen in the youngest cohorts.

The next section looks at available data on sun screens to see if they are a potential cause of the downturn. It is shown that sunscreens can be an explanation of the decline only under the hypothesis that it is control of intense exposure of skin which has not developed natural protection which is important if risk is to be reduced. Usage levels are too low for them to have been a factor if control of all exposure is necessary to reduce risk. The last section developes some of the potential benefits of a risk

communication strategy developed along the lines suggested by the results of the previous sections.

BACKGROUND

This section reviews some basic biology, some of the little that is known about how the skin develops natural protection, and some results about how exposure changes as a function of latitude, time of year, and time of day.

Melanoma arises in the melanocyte, the cell which produces melanin, the compound responsible for skin color.[10] The precise process by which the transformation to a tumor takes place is not known.[11] The tumor is normally highly antigenic--meaning the immune system will attack it--and one of the clinical markers for an early lesion is a red iritated area around the lesion.[12] Since UV radiation is known to suppress some aspects of the immune sytem, immune suppression via this route is thought to play some role in the disease.[13] However, this role remains to be worked out in detail. The tumor metastizes readily once it penetrates the surface of the skin and it is the metastases which are responsible for the mortality associated with melanoma.[14] On the other hand, five year survival rates for melanomas removed before the dermis has been invaded are about 95 percent.[15] Thus early diagnosis and removal are critical to effective treatment of the disease.

Incidence and death rates from melanoma have been growing very rapidly.[16] Figure 1 shows death and incidence rates for whites in the US. In 1984 total deaths from melanoma in the US were 5377. of these deaths 5264 were whites and 113 were non whites. Age

adjusted death rates were 3.11 for white males, 1.65 for white females, .37 for non-white males and .41 for non-white females. This is a world wide pattern, indicating that melanoma is primarily a disease of white populations. For non-whites, melanoma almost always arises in the non-pigmented portion of the body, either under the nails or on the soles of the feet.[17] Thus pigmentation is protective. This is true even within the white population, with southern Europeans such as Spanish and Portuguese much less likely to get melanoma than those of northern European origin.[18]

The hypotheses that melanoma might be solar related stems from the fact that non-melanoma skin cancers seem to be clearly sun related.[19] Non-melanoma cancers occur most frequently on the exposed portion of the body, and are much more frequent on those with lots of outdoor activity--thus they clearly are a function of lifetime exposure.[20] Melanoma, on the other hand does not follow this pattern.[21] Less exposed parts of the body, such as the trunk in males, and the legs in females, are the predominate place where melanoma is found. This clearly indicates the need for some modification of the solar hypothesis. The second problem stems from the results for Europe, which show that the expected decrease of melanoma incidence and death rates with latitude does not occur.[22] Rather rates are lower in southern Europe than in northern Europe. This may be due to the pigmentation variations discussed earlier. Later results in the paper on the possible role of recreation, occupation and residence patterns may also help explain the anomaly.

Existing results also point to the role of early exposure as being critical.[23,24] Again, the results are not unambiguous, and in some cases depend on quite small samples. Finally, due to the lack of an animal model, the exact portion of the spectrum responsible for carcinogenisis is not clear.[25] The hypothesis is that the UVB part of the spectrum is responsible, since this is the part of the spectrum where damage to DNA occurs. Due the lack of a widely distributed network of instrumentation capable of individual waveband measurement, there has been no confirmation of this by epidemiological studies. Thus the potential role of sunscreens as a protective device has been difficult to determine since the major chemicals are effective only in the UVB part of the spectrum.[26]

Exposure to the sun elicits the production of melanin and the development of a thicker stratum corneum, the outermost layer of cells on the skin.[27] Both of these factors reduce penetration of UV radiation to the growing layer of cells. While it is difficult to determine the exact extent of the protection induced by these factors, the tanning process does increase the length of time necessary to produce erythema(sunburn) by at least a factor of three.[28] Black skin reduces the level of radiation reaching the melanocyte by about a factor of 10.[29] Perhaps obviously, the incident angle of radiation is also very important, since radiation entering the skin at a sharp angle must travel much further before reaching the growing layer of cells. Thus, most work activities

expose substantially less of the skin to intense doses than do activities like sunbathing, where the body is prone.

Ultraviolet radiation present at ground level starts at about 290nm and increases by about 5 orders of magnitude in intensity by 325 nm. From this wavelength to 400 nm, the lower end of visible spectrum, radiation is rougly constant in intensity and varies in the same manner as visible light. The large variation in intensity between 290nm and 325nm is due to absortion by ozone in the stratosphere. Figure 2 shows variation in DNA weighted radiation by latitude for a clear day in the peak month of the year and for total radiation during the year. Note that there is little variation in peak values between the equator and 30 degrees. latitude. Figure 3 shows DNA and Erythema weighted radiation measures during the year for Washington DC. Note that DNA weighted radiation varies more than does erythema weighted radiation. Figure 4 shows variation during speak day in July. Note again that DNA radiation varies more during the day than does erythema. The relevance of these differences in behavior will become clear later in the paper.

PREVIOUS RESULTS FROM THIS PROJECT

The work already done on this data set bears on a number of the open questions discussed above. First, it shows that variations in intensity of ultraviolet radiation are associated with higher death rates.[30] A one percent increase in peak(clear summer day) DNA weighted radiation yields a .85 percent increase in the death rate for males and a .58 percent increase in the death

rate for females. Controls for socioeconomic variables do not affect the results while including the effect of ethnic origin reduces the responsiveness of death rates by about 20%.

The second area of work with this data set suggests that it is exposure in the UVB part of the spectrum which is responsible for the carcinogenisis.[31] The exposure measures used in previous epidemiology on melanoma have been simple latitude(which is a non-linear function of exposure as figure 2 illustrates), hours of sunlight, or an integrating meter(known as the Robertson Berger meter) which gives a single measure of UV radiation.[32] The exposure measure used in this study is developed from a model which incorporates satilite measures of ozone into a radiative transfer model to predict ground level UV radiation. These predictions can either be in the form of wavelength weighted measures where the weights are the inverse of the biological effectiveness of different wavelengths, or as individual waveband energies. In this particular work, individual waveband energies from 295-299 through 330-334 for a clear day in June were used as exposure measures. Table 1 presents the estimates for different wavebands. Deaths were modeled as a poisson process and estimation was done using iteratively reweighted least squares to get maximum liklyhood estimates. The results show a positive relationship between radiation below 320, with a negative and significant relationship above 330 for males. For females that pattern is similar, but the results are not significant above 330nm. Because of high correlations between different wavebands, it is not possible to

introduce more than two wavebands simultaneously into the equations. The second part of Table 1 shows the results using 295-299 and 300-304 as the short waveband with various wavebands used as the long waveband. This indicates the upper range for positive response to radiation lies at about 315 nm or at the upper end of the range where radiation damages DNA.

These results rely heavily on variations in specific parts of the spectrum. Since the model has only been tested with aggregate mesures produced by the Robertson-Berger meter, more work is needed to baseline the model. However, the overall pattern of variation is dependent only on variations in measured ozone and very basic radiative principles. Thus while there may be measurement error, it is unlikely to be systematic in nature, and thus, in this simple model, the expected result would be to bias the estimated coefficients toward zero.

The third set of analyses done with this data look at cohort experiences.[33] As seen in Figure 5, there is a very systematic structure to a plot of the log of the national cohort death rates against age. Cohorts are defined as those who are 0 to 4 years of age for a five calender year interval. This results in a median birth year equal to the initial calender year of the period. This definition was required because death data were only available in five year age groups in the source data set. The labels on the plot refer to the median birth year for each cohort. The parallel slopes of the cohort death rate curves above the age of thirty suggest that it is early exposure which is critical to the

potential risk. Statistical analysis confirms that the curves above the age of 30 have equal slope. For men this slope is 7 percent per year and for women it is five percent per year. As Figure 5 shows, there is no slope to the death rate experience before age 10. Clinical experience indicates these deaths are due to congenital nevi.[34] Therefore, it seems reasonable to assume that the rate for 0 to 9 year olds is constant, and all the variation in cohort risk is due to variation in how the death rate changes between age 10 and age 30. Table 2 presents estimates for a model which includes DNA weighted exposure, individual cohort estimates for $7 < Age < 32$, and a common age effect above age 32. These results suggest that variations in some aspect of exposure across time for the age group less than 30 are at the root of the varying coefficients for the cohort specific age variable.

Using a much simpler procedure, estimates at age specific rates at age 32 can be made for chorts born between 1865 and 1970. For the 35 years of data available, average ratios for each five year differential are computed. These averages are used to extrapolate from the nearest available death rate to the age 32 death rate. Table 3 gives the results of these forecasts for each birth cohort. White males show marginally greater than a ten to one variation while white females show about a five to one variation. Interestingly, there is a predicted downturn in the age specific rates for cohorts born after 1950. As seen in figures 5 and 6, these reductions are already seen in these cohorts at younger ages.[35] Next to the differential rates for blacks and

whites, this is the largest variation seen in experience with melanoma. Thus any explanation of melanoma aetiology must deal with this experience. The next section of the paper looks at some potential explanations for these large cohort effects.

COHORT VARIATIONS IN DEATH RATES

Given the small number of degrees of freedom across cohorts and the very limited quantity and quality of data on recreation in particular, the analysis in this section is more qualitative in nature that the analysis in the previous sections. The essential question to be addressed is what changes have occurred in exposure habits and opportunities between 1880-84, when the oldest cohort in the study was 15-19 years old and the 1980-84 period, when the 1965 cohort was 15 to 19 years of age. There are a number of hypothesis which could be suggested for the variation across this period of time. Here only solar related hypotheses are considered since there is little indication in the literature of any other cofactor besides genetic predisposition as a potential cause of melanoma. (This is not to say one might not exist--but only that a creditable one has not been found so far).

The first potential hypothesis is that changes in place of residence during the critical exposure years might have changed so that average intensity of exposure is higher. However, as Table 4 shows, DNA relevant radiation weighted by state populations between 15 and 24 for every five years between 1890 and 1985 increases by only 2.8 percent in intensity(average exposure in 1980 is 3.25). Since this would amount to only an 2.5 percent change

in risk at age 32 for males and a 1.65 percent change in risk at age 32 for females, this does not explain the very large changes in lifetime risk seen in Table 4.

Likewise, occupational exposure is not the explanation. The two major occupation groups with extensive sun exposure are farming and construction. As Table 5 shows, these have fallen sharply in relative size, and even in absolute size during the 1880-1985 period. Also occupation is less apt to be a risk factor for those under the age of 20 since labor force participation rates are relatively low and have been quite static in the 50 to 60 percent range for white males between 14 and 19 and between 20 and 30 percent for white females in the same age group.

A third potential hypothesis centers around outdoor recreational exposure. This can at best be a partial explanation of the changes in melanoma risk. Around 1900, forty percent of the population lived on farms and participation in outdoor recreation was about 4 hours per capita per year(see Table 7), while the lifetime risk of death from melanoma was only about 1 in 1000 for both males and females. In 1960, eight percent of the population lived on farms, per capita participation in outdoor recreation had risen to almost 120 hours, and the lifetime risk of death from melanoma had reached 6 per thousand for males and 2.7 per thousand for females. From 1960 to 1985, farm population fell to about 2.5 percent of total population, per capital outdoor recreation hours by 2 and one/half fold, but the risk of melanoma has decreased. While the results between 1900 and 1960 are suggestive of a role

for outdoor recreation, the 1960 to 1985 results suggest(as always with melanoma it seems) that if there is a role for recreational exposure it is not a simple one.

One can make sense of the role of recreation if what matters is not the extent of participation, but simply participating at all. Under this hypothesis one would expect to see a stabilization in participation rates in sun intensive activities beginning in the sixties. Unfortunately, data on a comparative basis does not exist. What can be examined is a number of proxy variables for participation. One proxy for recreation behavior is the percent of the labor force not at work due to vacation(see Table 8). While comparable data is not available before World War 2, data given in Clawson and Knetch indicate weeks of vacation per worker rose from .37 in 1929 to 1.09 in 1959, suggesting percent participation rose during the 1929-1946 interval also.

A second indicator of percent participation comes from noting that over ninety percent of outdoor recreation involves automotive **transportation.**[xx] Assuming that most recreational activity involving automotive transportation is family oriented, the critical variable controlling access to outdoor recreation is household motor vehicle ownership. Table 9 gives this data for the post war period. Table 10 extends this back before WW II as mean vehicles per household. Comparison with Table 9 indicates that mean vehicles oer household is roughly double the percentage of households owning at least one car. What is clearly interesting

about this variable is the apparent saturation on a per household basis which occurs in the early 1960's.

These two variables suggest that a case can be made that the breadth of participation in outdoor activity stablized in the 1960's. Several surveys of participation in outdoor activities were done between 1962 and 1982-83. While summary results from these surveys are not in a format that makes comparison across time possible, the latest survey(1982-83) does indicate that all but 11 percent of the general population participate in some form of outdoor recreation. For those between 12 and 24, all but 3 percent of the population participate. The next step in this process is to get the source level documents and see if a more coherent picture can be developed.

The other aspect of changes in recreational exposure which is important to understand is that activities associated with intense exposure have increased over time. Swimming, especially sunbathing, is typically associated with more intense exposure than hiking or bicycling. Further, in the northern part of the country, the outdoor swimming season does not begin until the intensity level is within ten percent of the peak level it will achieve during the year. One illustration of the increase in intensity is that the number of muncipal swimming facilities per capita increased more than seven fold between 1910 and 1965.

The issue of when expsoure begins, alluded to in the previous paragraph, is also important. There have been very significant changes in the time at which exosure begins for the critical age

groups. In the 1880's, they typical student attended school for only 80 days a year, and stoped school after the eighth grade. Today, the typical student attends school twice as long and in excess of 95 percent of the 5-17 year old population is in school. Given the shortness of the typical school year before 1900, we can suppose that the real pattern for farm children, especially those in the early teens, was to be outside helping with farm work beginning in the spring and continuing through the fall. This is not a pattern of limited sun exposure. It is a pattern which leads to the development of a tan prior to the period of peak intensity. As Table 1b shows, if the participation in farm work begins in March or April, the exposures levels are much lower than those found beginning in late May or June, the typical time at which school closes in the modern era and outdoor recreation starts.

Thus we have a potential hypothesis explaining the increase in melanoma as a function increase in the effective intensity at the time when sun exposure begins for the season. Under this hypothesis, the stabilization of rates occurring in the 1930's to 1950 for females and in the 1950's for males is explained by stabilization in the percent of the population getting intense exposure. The large increase in recreation behavior since the sixties is one of more extensive participation by each individual, rather than a broader participation. If this is in fact the case, and more work is needed on this score, then a consistent pattern can be found which explains the growth of melanoma by an increase in the effective intensity of radiation brought about by changes

in education and work patterns which delay the onset of outdoor exposure until the period of peak insolation, and the spread of activities such as swimming, which expose much of the body to sunlight, especially parts of the body which rarely receive any prior exposure.

This would explain the markedly lower rates seen for the head neck and hands for melanoma, since these parts of the body are exposed year round and thus have always developed some level of natural protection. It does not however, explain the decrease in melanoma rates seen in cohorts born since the 1950's. One possible explanation, consistent with the solar hypothesis, is that sun screens have played a role. This is discussed in the next section.

SUNSCREENS

Under the intensity hypothesis, to be effective in reducing risk, sunscreens do not have to be used all the time, but only during the period of initial exposure. The question is whether the total use Of Sunscreens, given in Table 12, is sufficiently high to have possible been effective in reducing risk. To provide adequate protection at the rated level, about one ounce of product is required.[xx] Typical applications to the entire body seem to be at about half this rate.[xx] This rate is still enough to produce a very significant reduction in risk. Thus the actual number of applications available is about twice the number of ounces sold. This yields about three applications per individual, which is probably a minimum level of protection for one day on the beach, but not enough to get all potentially exposed individual through

the period of developing a tan without getting an intense dose. Thus it is unlikely that sun screens are the potential explanation for the declining risk seen in the younger age cohorts. Only if all sunscreen use were concentrated in the younger cohorts would this be possible. In fact, anecdotal evidence suggests it is the younger cohorts which are least apt to use sunscreens. Thus some other explanation for the decline must be sought. This does not mean that a policy of increased use of sun screens would be ineffective. The potential of such a policy is discussed in the next section.

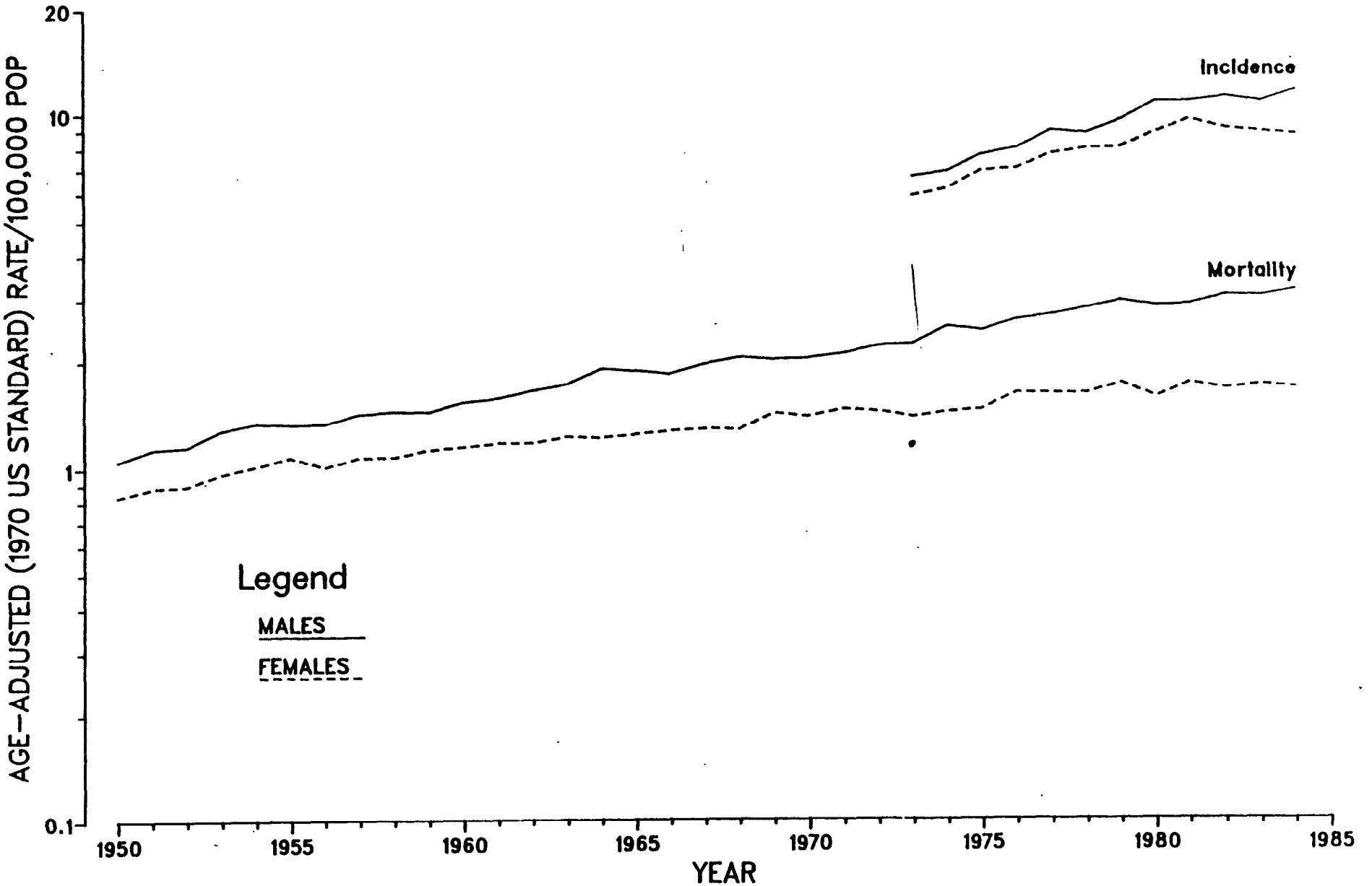
FOUNDATION OF A RISK ASSESSMENT POLICY

The intensity hypothesis suggests that a policy is possible which might be very effective in reducing melanoma. The primary goal of the policy would be to limit exposure very carefully during the period before the skin has a chance to develop its natural defenses of thickenign of the stratum corneum and tanning. Since these processes both take time, this would imply either the careful use of sunscreens or a significant limitation on activity during a vacation taken by somebody who starts exposure when natural intensity levels are high or travels to a sunny area during the winter.

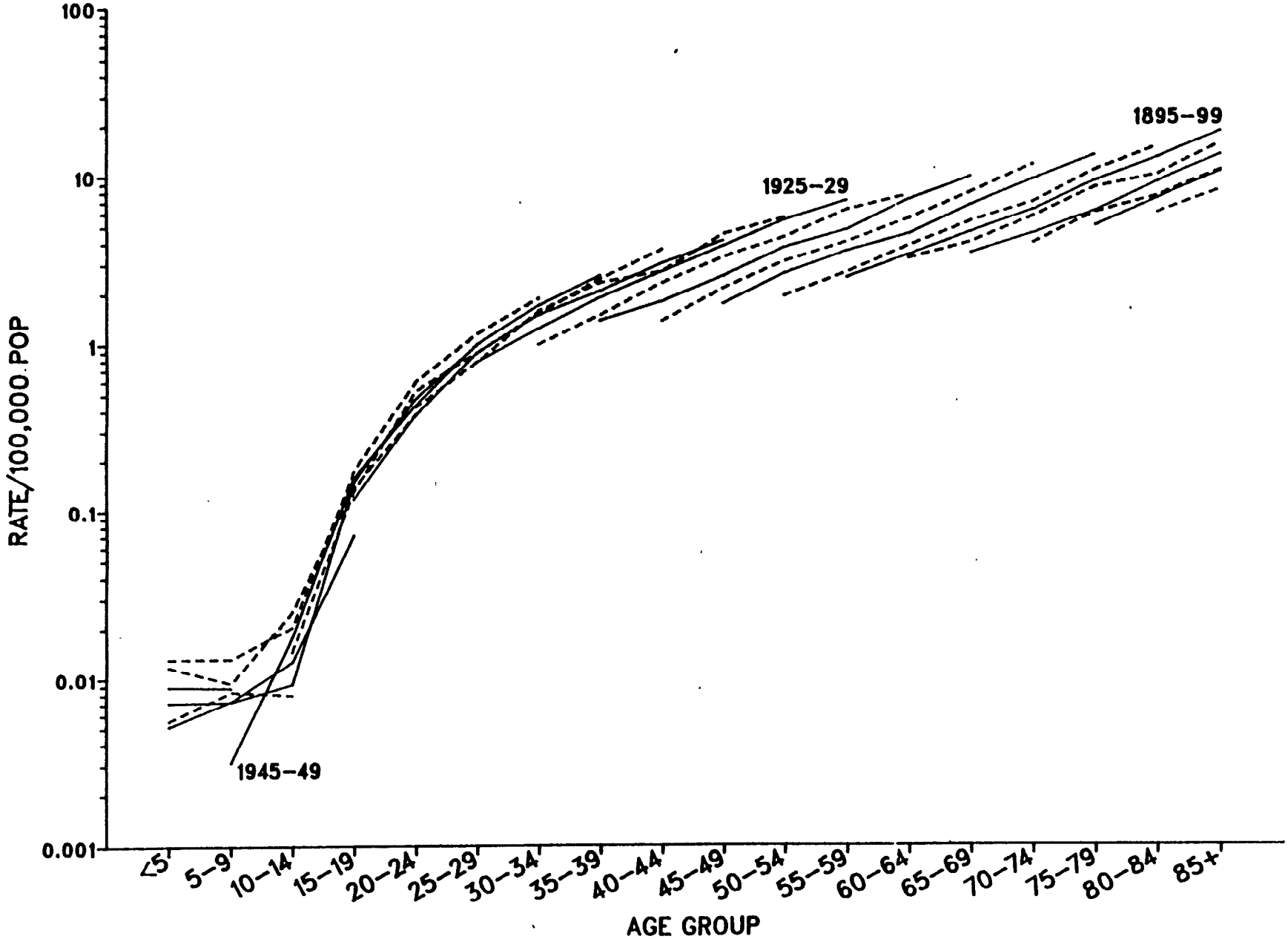
How much might such a prevention program be worth. No strategy could probably return us to the results of the 1900 era. There is simply too much intense radiation present in modern recreational activities and use of sun screens is unlikely to be universal. However, it might be reasonable to reduce risk by 60

percent. The results of a policy with this level of effectiveness are illustrated in Table 12. Since we have a fairly detailed sense of the death rate pattern for melanoma, the table looks at years of life saved rather than reductions in mortality. The figures are for a cohort group of 100,000. The total reduction in melanoma mortality, in a given year, under steady state cohort behavior, would be 368 lives per 100,000 males, and 162 lives per hundred thousand females. At currently typical white birth cohort sizes of about 1.75 million each for males and females, this yields a total reduction of better than 9000 melanoma related deaths. Total associated incidence would be about four times these levels, giving reduced incidence of about 36,000 cases. It should be emphasized that these are long run numbers and do not take account of whatever is currently acting to reduce death rates. They do suggest that a policy to moderate sun exposure habits has a very high potential public health payoff.

Skin Melanoma Trends Among Whites in the United States



Skin Melanoma Mortality by Birth Cohort
Among WHITE MALES



Skin Melanoma Mortality by Birth Cohort
Among WHITE FEMALES

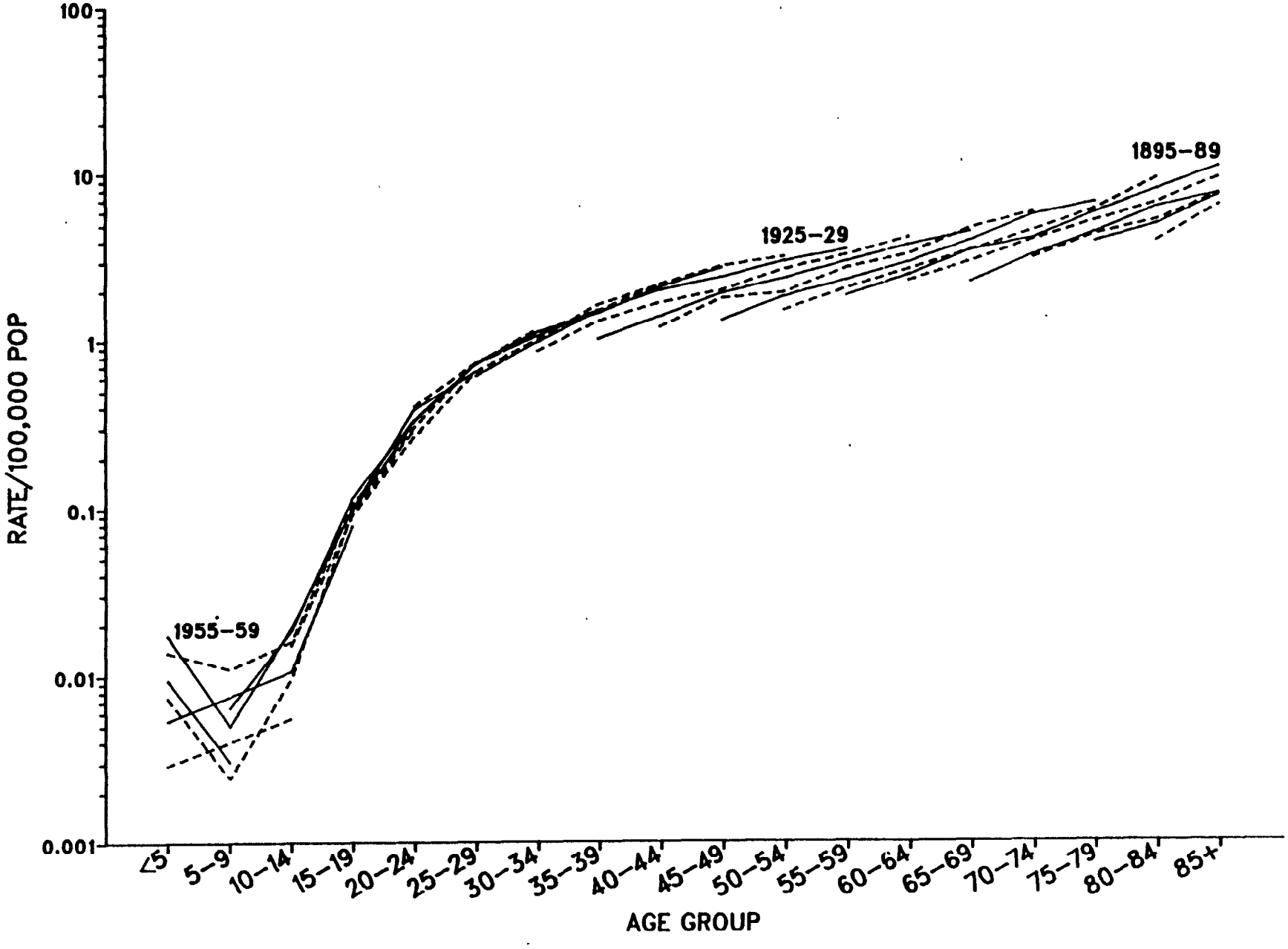


Table 1a

Ultraviolet Radiation Variation by Latitude

Latitude	Clear Day DNA Weighted	Annual DNA Weighted
0	5.45	1162.5
10	5.39	1181.6
20	4.59	989.1
30	4.14	658.0
40	3.31	370.7
50	2.33	204.0
60	1.67	124.3

Table 1b

Clear Day UV Radiation by Month

Month	DNA Weighted	Erythema Weighted
Jan 15	.25	31.0
Feb 15	.50	57.9
Mar 15	1.01	108.7
Apr 15	1.85	186.8
May 15	2.51	246.5
Jun 15	3.14	299.6
Jul 15	3.40	318.3
Aug 15	2.91	274.6
Sep 15	2.03	196.2
Oct 15	1.09	110.9
Nov 15	.46	51.2
Dec 15	.24	28.9

Source: Model based estimates using satelite data on ozone. Units are not comparable.

Table 1c

UV Radiation on July 15(Clear Day)

Time of Day	DNA Weighted	Erythema Weighted
6-6:30 am	.0055	.78
6:30-7 am	.0129	1.66
7-7:30 am	.0259	3.06
7:30-8 am	.0456	5.03
8-8:30 am	.0723	7.55
8:30-9 am	.1050	10.53
9-9:30 am	.1419	13.77
9:30-10 am	.1800	17.05
10-10:30 am	.2162	20.10
10:30-11 am	.2469	22.66
11-11:30 am	.2693	24.51
11:30-12 am	.2811	25.48
12-12:30 pm	.2811	25.48
12:30-1 pm	.2693	24.51
1-1:30 pm	.2469	22.66
1:30-2 pm	.2162	20.10
2-2:30 pm	.1800	17.05
2:30-3 pm	.1419	13.77
3-3:30 pm	.1050	10.53
3:30-4 pm	.0723	7.55
4-4:30 pm	.0456	5.03
4:30-5 pm	.0259	3.06
5-5:30 pm	.0129	1.66
5:30-6 pm	.0055	.78
6-6:30 pm	.0020	.30
6:30-7 pm	.0006	.09

All radiation values from radiative transfer model incorporating satelite measurements of ozone.

Table 2a

Wavelength Specific Estimates of Exposure Effects*

Males

Wavelength	Coefficient	Standard Error
295-299nm	.142	.0104
300-304nm	.148	.0108
305-309nm	.153	.0115
310-314nm	.145	.0125
315-319nm	.112	.0132
320-324nm	.055	.0139
325-329nm	.0014	.0143
330-334nm	-.028	.0143
335-339nm	-.044	.0136
355-359nm	-.064	.0138

Females

295-299nm	.0817	.0123
300-304nm	.0871	.0128
305-309nm	.0923	.0135
310-314nm	.0912	.0144
315-319nm	.0765	.0152
320-324nm	.0458	.0157
325-329nm	.0159	.0158
330-334nm	-.00090	.0158
335-339nm	-.0106	.0149
355-359nm	-.0204	.0152

Table 2b

Multiple Waveband

	Male	Female
295-299nm	.151	.0709
	(.0182)	(.0219)
315-319nm	-.0255	.0197
	(.0197)	(.0225)
295-299nm	.150	.0804
	(.0114)	(.0136)
320-324nm	-.0298	.0012
	(.0152)	(.0176)
300-304nm	.166	.0908
	(.0124)	(.0147)
320-324nm	-.0472	-.0091
	(.0158)	(.0182)

* All units have been converted to standard deviation form so that coefficients can be directly compared.

Table 3

Age and Exposure Model Coefficients

Variable	Male		Female	
	Coef	St.Dev.	Coef.	St. Dev.
Constant	-3.99	.118	-3.90	.117
DNA expos.	.263	.0125	.188	.0137
Age65	.0730	.0430	.074	.0405
Age60	.146	.0153	.126	.0162
Age55	.154	.00859	.143	.00874
Age50	.155	.00583	.142	.00597
Age45	.150	.00523	.140	.00528
Age40	.149	.00493	.138	.00496
Age35	.143	.00480	.139	.00476
Age30	.143	.00469	.139	.00465
Age25	.139	.00463	.132	.00460
Age20	.131	.00462	.126	.00458
Age15	.125	.00463	.120	.00460
Age10	.117	.00467	.117	.00463
Age05	.110	.00469	.111	.00466
Age00	.102	.00473	.107	.00471
Age95	.0962	.00478	.105	.00475
Age90	.0921	.00480	.100	.00478
Age85	.0837	.00483	.0928	.00482
Age80	.0806	.00487	.0920	.00486
Age>32	.0653	.000817	.0538	.000882
Sum of Squares				
Regression	4273.7		2655.3	
Error	200.7		153.0	
Total	4474.4		2808.4	
About Mean	4199.7		2577.1	

The agexx variables denote a variable which for cohort xx is

0	if Age < 7
age - 7	if 7 < Age < 32
25	if Age > 32

and 0 for all other cohorts. Cohorts are denoted by the last two years of their median birth date and run from 1965 back to 1880. The Age>32 variable is 0 if Age < 32 and Age - 32 otherwise.

Table 4

Predicted Rates at Age 32

Median Birth Year	White Males	White Females
1865	.146	.238
1870	.194	.234
1875	.222	.311
1880	.257	.330
1885	.309	.313
1890	.398	.424
1895	.419	.441
1900	.438	.460
1905	.553	.494
1910	.643	.593
1915	.907	.693
1920*	.986	.857
1925*	1.224	.969
1930*	1.555	1.109
1935*	1.482	1.116
1940*	1.509	.998
1945*	1.690	1.051
1950*	1.866	1.155
1955	1.690	.998
1960	1.330	.954
1965	.800	.807
1970	.795	.395

* denotes observed value for that cohort. Other values are predicted from the nearest observed value and the average ratio between age 32 and the observed value at that age.

Table 5

Population(Age 15-24) Weighted Measures of Exposure

Year	DNA Exposure
1890	3.185
1900	3.201
1910	3.194
1920	3.205
1930	3.212
1940	3.212
1950	3.225
1960	3.239
1970	3.238
1980	3.263
1985	3.276

* Data on population from US Historical Statistics and various issues of US Statistical Abstract. UV measures are mean of SMSA spectific measures within each state.

Table 6

Farming and Construction Employment

Year	Total	Farming	Construction	Percent
1880	17390	8920	900	56.5
1890	23320	9690	1510	48.0
1900	29070	11680	1665	45.9
1910	37480	11770	1949	36.6
1920	41610	10790	1233	28.9
1930	48830	10560	1988	25.7
1940	56290	9575	1876	20.3
1950	63377	9926	2364	19.4
1960	71489	7057	2926	14.0
1970	84889	4596	3588	9.6
1980	108544	3705	4346	7.4
1985	117167	2941	4673	6.5

* Data from 1950 are not strictly comparable to earlier data. Data from 1880 to 1940 are from US Historical Statistics, US Dept of Commerce, Washington DC 1975(Series D167, D170 and D173). Data from 1950 to 1985 are from Economic Report of the President, Council of Economic Advisors, Washington, DC, 1989(Tables B32, B43, and B98).

Table 7

Per Capita Hours of Recreational Activity

Year	Hours
1900	4
1910	7
1920	20
1930	43
1940	59
1950	80
1960	116
1970	211
1980	272
1985	304

*Data through 1960 are adapted from Clawson and Knetch, The Economics of Outdoor Recreation. From 1970 to 1985, they are extended by computing an index based on visits to National Parks, National Forests, State Parks, Personal Consumption Expenditures for Gardening, and Travel to the Carribean and South America.

Table 8

Percent Participating in Vacation
Not at Work by Reason of Vacation
(annual average, 1000's)

Year	On Vacation	Percent
1985	3338	34.2
1980	3320	36.7
1975	2815	35.4
1970	2341	33.1
1965		
1960	1576	26.5
1955	1268	22.7
1950	1137	21.5
1946	662	13.8

* Percent assumes everybody counted during the year as being not at work due to vacation is distinct and multiplys not at work by 12 to get an estimate of total fraction of the work force which takes a vacation.

Table 9

Household Ownership of Motor Vehicles

Year	Total	Percent One Car	Percent Two or More
1948	54		
1950	59	52	7
1955	70	60	10
1960	77	62	15
1965	79	55	24
1970	82	54	28
1977	84	47.5	36.5

Source: US Historical Statistics 1948-1970, Motor Vehicle Facts and Figures, 1978.

Table 10

Access to Motor Vehicles

Year	Cars (1,000's)	Total (1,000's)	Per Capita	House-holds (1,000's)	Per HH
1890	0	0	0	12690	0.0
1895	0	0	0	14341	0.0
1900	8	8	0.00011	15992	0.0005
1905	77	79	0.00094	17939	0.0044
1910	458	469	0.0051	20183	0.023
1915	2332	2491	0.025	22501	0.11
1920	8132	9239	0.087	24467	0.38
1925	17440	19941	0.17	27540	0.72
1930	22973	26532	0.22	29997	0.88
1935	22495	26230	0.21	31892	0.82
1940	27372	32035	0.24	35153	0.91
1945	25695	30638	0.22	37503	0.82
1950	40191	48567	0.32	43554	1.12
1955	51961	61949	0.37	47874	1.29
1960	61420	72887	0040	52799	1.38
1965	74909	89090	0.46	57251	1.56
1970	88775	106808	0.52	63401	1.68
1975	106077	130919	0.61	71120	1.84
1980	120866	153358	0.67	80776	1.90
1985	129329	167342	0.70	86789	1.93

Table 11

Sales of Sun Protection Products
(Real 1988 $, 1,000,000's)

Year	Total		SPF > 8	
	Sales	Ounces	Sales	Ounces
1960	85.6	57.1		
1965	115.8	77.2		
1970	155.3	103.6		
1975	193.9	129.3		
1980	238.9	159.3	80.0	53.3
1984	249.0	189.5	103.3	68.9

Source: I am indebted to Jim Murdoch and Mark Thayer for providing the original data.

Table 12

Potential Mortality Impacts
of Reducing Acute Exposure

Males-1940 Birth Cohort

Age	Baseline Mortality	Reduced Mortality	Difference	Expected Lifetime	Total Add. Yrs.
0-4	.28	.07	.21	70.7	14.85
5-9	.26	.07	.19	65.8	12.50
10-14	.51	.20	.31	60.9	18.88
15-19	1.04	.42	.62	56.1	35.01
20-24	2.05	.82	1.23	51.4	63.22
25-29	4.06	1.63	2.44	46.8	114.00
30-34	8.00	3.20	4.80	42.2	202.56
35-39	11.27	4.51	6.76	37.5	253.58
40-44	15.77	6.31	9.46	32.9	311.30
45-49	22.39	8.96	13.43	28.4	381.53
50-54	30.93	12.37	18.56	24.2	449.10
55-59	42.02	16.81	25.21	20.2	509.28
60-64	55.68	22.27	33.41	16.6	554.57
65-69	71.09	28.44	42.65	13.3	567.30
70-74	85.72	34.29	51043	10.5	540.04
75-79	95.00	38.00	57.00	8.0	456.00
80-84	93.35	37.34	56.01	6.0	336.06
85+	75.98	30.39	45.59	4.5	205.15
Total	615	247			5025

Table 12 Continued

Females-1940 Birth Cohort

Age	Baseline Mortality	Reduced Mortality	Difference	Expected Lifetime	Total Add. Yrs.
0-4	.26	.10	.16	77.4	12.07
5-9	.24	.10	.14	72.5	10.44
10-14	.45	.18	.27	67.6	18.25
15-19	.85	.34	.51	62.7	31.98
20-24	1.57	.63	.94	57.8	54.44
25-29	2.92	1.17	1.75	53.0	92.86
30-34	5.40	2.16	3.24	48.1	155.84
35-39	6.95	2.78	4.17	43.3	180.56
40-44	8.89	3.56	5.33	38.5	205.36
45-49	11.51	4.60	6.91	33.9	234.11
50-54	14.63	5.85	8.78	29.4	258.07
55-59	18.43	7.37	11.06	25.0	276.45
60-64	22.95	9.18	13.77	21.0	289.17
65-69	28.04	11.22	15.12	17.2	289.37
70-74	33.30	13.32	19.98	13.6	271.73
75-79	37.81	15.12	22.69	10.5	238.20
80-84	39.81	15.92	23.89	7.7	183.92
85+	36.05	14.42	21.63	5.6	121.13
Total	270	108			2924

FOOTNOTES

Forthcoming at the Conference

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Two basic citations--detailed bibliography available at the conference.

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Acute Health and Variable Air Pollutants

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I. Introduction

Previous epidemiological studies [4,5,6,8] have established an empirical relationship between measures of urban air pollution (the dose) and human illness (the response). The results from these studies are interesting to policy analysts because they can, in principle, be used to predict the impacts of proposed air pollution control policies on urban populations. These aggregate dose-response predictions are credible to the extent that they seem to confirm the association between air pollutants and illness that has been found in clinical studies [2,7]. Nevertheless, the sensitivity or robustness of the predictions from the estimated dose-response functions to alternative specifications, datasets, and estimation strategies remains an important issue [2].

This paper addresses two methodological issues in estimating air pollution dose-response functions. Both concern accuracy in measuring the air pollution dose.

The first involves the intracity variation in the pollution data and the location of respondents. Previous studies have not assigned respondents different location codes over the day. Yet many people, especially those working, get a different dose during the day, when compared to the evening when they are at home. The dataset analyzed here facilitates a work and home assignment of air pollution to individuals within a city. This allows us to compare the responses of individuals to air pollution at their workplace with their responses to air

pollution at home. By modeling more of the variation in the pollution data, we measure the "real-world" dose; hopefully, improving the accuracy of the estimates of the influence of air pollution on human health.

The second issue concerns the intralocation variation in air pollution. Since air pollution in an urban area can vary from hour to hour, we hypothesize that the dose is more appropriately modeled as variable over a day. Even when an individual does not change locations, he or she will experience different doses as the air pollution varies from hour to hour. Therefore, the air pollution dose depends on where and when a person is exposed. The air pollution doses used in previous studies have been based on a periodic (either one year or two weeks) average. By averaging the pollution data, the intraday variation in the data, which may influence human health, has been ignored. This can cause specification error bias, owing to left out and incorrectly measured variables in the dose-response function.

The remainder of this paper is organized into four sections. In Section II, we present a brief review of the relevant literature on estimating the relationship between air pollution and human morbidity. The empirical models, data, and basic estimation methods are described in Section III. The results are presented in Section IV, while the last section contains concluding remarks.

II. Air Pollution and Morbidity: Previous Studies

The design of this paper is most closely related to the

studies by Ostro [4,5], Hausman et al. [3], and Portney and Mullahy [6]. These authors use the Health Interview Survey (HIS), an annual health survey of people in various locations throughout the U.S., to study the empirical relationship between air pollution and human morbidity. Morbidity is measured by a variable that reflects the changes in the normal activities, owing to health impairments, of the survey respondents during the 2-week recall period of the HIS. Several pollutants are analyzed, including measures of the atmospheric concentrations of total suspended particulate (TSP), ozone, fine particulate, and sulfates. In addition to including several socio-economic and weather measures in their models; these authors examine numerous subsamples based on sex, working status, and smoking status, attempting to hold constant as many confounding influences as possible.

In Ostro [4], the variation in the air pollution data comes from the pooling of respondents from different cities. Doses were measured by the annual average of total suspended particulate (TSP) and sulfates (SO4) and ignore the intrayear and intraday variation in the pollution data. The morbidity measures reflect the number of “work loss days” (WLD) and the number of “restricted activity days” (RAD) that survey respondents reported during the two-week recall period. The TSP term is significant and has the expected relationship to RAD and WLD. The SO4 term is not significant, which may not be surprising since SO4 is more localized than TSP.

In his follow-up piece, Ostro [5] uses a Poisson distribution to model the relationship between the number of RADs and the contemporaneous (with the survey) two-week average of fine particulate. Fine particulate are estimated from airport visibility and TSP data. The two-week average of fine particulate is significant over several different samples and years.

Hausman et al [3] concentrate on WLD and specifically control for intrayear variation in pollution. Additionally, they estimate models with alternative lags of the two-week (in contrast to the annual) average of TSP, although no formal tests to choose among the specifications are presented. Like the Ostro studies, intracity variation in the pollution exposure (within a period) is ignored and the SO4 measure is not significant. Hausman et al provide some empirical support for the Poisson specification; i.e., the pollution coefficients were robust when the Poisson assumption that the variance equal the mean was relaxed and when a fixed effects model was estimated.

Portney and Mullahy [6] estimate a Poisson model with the number of respiratory related RAD as the dependent variable and various measures of ozone and sulfates for the exposure variables. Portney and Mullahy's ozone exposure measures are probably better suited to test acute health effects because they average over the daily maximum of ozone during the two-week recall period. Moreover, by matching respondents to the pollution monitoring stations closest to their census tract (and

within a 10 and a 20 mile radius of their census tract), Portney and Mullahy analyze some of the within city variation in the pollution data. They find that the estimates for the ozone coefficients do not vary greatly among these different assignment strategies, meaning that the intracity variation is not empirically important in their data. As in the aforementioned studies, sulfates do not perform in an a priori expected manner.[1]

III. Methodology and Data

The methodology and data used in this study were constructed in order to examine the robustness of acute health predictions. In particular, we propose to compare the predicted health responses from a "traditional" specification to specifications where the pollution measures and assignments more accurately reflect real world exposures. This comparison exercise will provide information for policy makers as well as future research efforts.

Define the following notation:

- H_{it} = the health response of individual i in time period t.
- X_i = a vector of individual specific covariates.
- W_t = the weather in time t.
- $POL_{it}(L)$ = the pollution exposure experienced by i in time period t. The exposure is a function of i s location (L) over the time period.

Then,

$$H_{it} = f(X_i, W_t, POL_{it}(L)) \qquad (1)$$

is a hypothesized dose-response function.

To estimate a model like (1), requires data on H_{it}, X_i, W_t, and $POL_{it}(L)$ and a functional form for the model. The necessary data were obtained from a health survey, the Weekly Weather and Crop Bulletin, and the SAROAD system data tapes. The functional form for the model was specified to be consistent with previous studies.

The health survey data

During 1978-1980, Geomet Technologies, Inc. administered a health survey to the members of 2,594 households in the greater St. Louis area. Households were enrolled in the survey in groups of about eighty per week beginning June 4, 1978 and ending May 27, 1979. The respondents maintained daily logs of their activities, locations, restrictions in activities, and the reasons for any restrictions in activities. The logs were kept over four two-week periods; thus, the dataset includes the restrictions on activities and the locations for each respondent for 56 days.

The structure of the survey also facilitated the collection of extensive data on socioeconomic conditions, lifestyle choices, work environment, medical care, and health.[2] A complete description of the data and the datafiles are available from the authors upon request.

The appropriate measure of the health response depends on the focus of the study. Here, we are particularly interested in acute respiratory health responses. As an empirical measure of

H_{it}, we used the number of RADs reported by the respondent in the time period, owing to a respiratory disorder or symptom ($NRRAD_t$). Given this type of limited dependent variable, a reparameterization of the Poisson distribution is a particularly attractive statistical model for equation (1).

As shown elsewhere [6], the expected value of $NRRAD_t$ under the Poisson model is given by

$$E(NRRAD_t) = \exp(X_i\beta + W_{it}\gamma + POL_{it}(L)\delta) \quad (2)$$

where the β, γ, and δ represent parameter vectors that are estimated via maximum likelihood methods. Using equation (2), a prediction for a small change in a $POL_{it}(L)$ variable (or any other) is a straightforward computation.

The variables in X_i should include measures on i's age, income, living arrangements, working conditions, personal health habits, and personal health status. Since an incorrect specification of the X_i could bias the estimates of the relationship between H_{it} and $POL_{it}(L)$, we included several covariates. Moreover, the data were limited to people between the ages of 16 and 65 who are non-smokers and working outside of the homes

A brief description and summary statistics of the X_i covariates, the weather covariates, and NRRAD are presented in Table 1.

Pollution Data

The pollution data were obtained from the U.S. Environmental Protection Agency's SAROAD system. The data tapes contain hourly

observations, collected at 14 monitoring sites, on numerous pollutants in the St. Louis Air Quality Control Region.[4] The pollution data were matched to the survey respondents by time, as described below, and location vis-a-vis the monitoring stations. The respondents averaged about three miles from a monitoring site. The pollutants analyzed here, ozone and sulfur dioxide (SO2), were chosen for two reasons. First, the data for these two pollutants were collected at all of the monitoring stations over the time period of the health survey. For the other air pollution measures, for example, total suspended particulate and NO2, the data are not available for several weeks during the survey period or they were not collected at each site. Second, SO2 tends to be more localized than ozone. This contrasting nature of the two pollutants provides a natural “laboratory” for measuring the appropriateness of our measures and assignments of pollution.

The pollution measures differ from the X_i and the W_t because an individual's exposure to pollution is not constant over t. Pollution exposure can varyas individuals change locations over the day. Even when an individual is stationary, their exposure changes as the pollution varies over the course of the day.[5]

In defining the measures of air pollution dose, our objective was to preserve as much variation in the pollution data as possible. The format of the health survey means that, for each enrollment week, there are four associated two-week periods. Since we used the survey data from weeks 1 through 41, there are

164 two-week periods. However, within each two-week period, we grouped the data into “day-time” observations (the hours 11:00 am through 5:00 pm) and “night-time” observations (the hours 5:00 am through 10:00 am and 6:00 pm through 12:00 pm). The pollution data were, therefore, initially grouped into 328 subperiods. Each day-time subperiod contains 98 observations, while the night-time subperiods consist of 182 observations.

For each subperiod, we computed the following sets of parameters:

(i) The mean and standard deviation. If the data are normally distributed, then these parameters fully describe the distribution of the pollution.

(ii) The mean and standard deviation of the natural logarithm of the data. If the data are lognormally distributed, which may be more plausible than normality, then these parameters characterize the distribution of the dose.

Also, to facilitate a comparison with previous models, the two-week mean and the average over the daily maximums were computed.

Two methods were developed for investigating the sensitivity of the dose-response function to the individual’s pollution exposure. Both address the variability in air pollution doses.

<u>The impact of locations changes on pollution measures</u>

For an individual who lives in one location and works in another, we are uncertain about the correct assignment of the pollution dose. With the data analyzed here, each respondent has two location codes; a home code and work code. The home location

pollution, the work location pollution, or some combination of the two could seemingly be used to assign pollution exposures to the individuals. Moreover, the pollution data reflect different times of the day; thus, the home code may be more appropriate for night-time exposures and the work code more appropriate for day-time exposures. Since we are uncertain about the correct assignment, one possibility is to let the data determine it.

Let θ_1 be the fraction of the total exposure time to air pollution experienced during the day at work. Similarly, let θ_2 be the fraction experienced at home during, our definition of, the night-time. Finally, let θ_1 be the fraction of exposure time experienced at home during the day. We assume that $\theta_1 + \theta_2 + \theta_3 = 1$, implying that all of the exposure is experienced in the manner hypothesized.

The θ's, if assumed to be unknown, can be estimated given some criterion. the correct mean exposure experience by a respondent is a weighted average of the means at each location for each time period, where the weights are the θ's. Let

WDPOLMU = the mean of pollution calculated from the day-time data at the work location code,

DPOLMU = the mean pollution calculated from the day-time data at the home location code, and

NPOLMU = the mean pollution calculated from the night-time data at the home location code.

Then,

$$\text{POLAVE} = \theta_1\text{WDPOLMU} + \theta_2\text{DPOLMU} + \theta_3\text{NPOLMU} \qquad (2)$$

is the weighted average pollution experienced by the respondent. We use a grid search over the θ's to find the set that maximizes the likelihood function.

As reference points, we alternatingly let each of the θ's have a value of 1. Additionally, we assumed 112 hours of exposure per week; 40 at work, 56 at home during the night-time hours, and 16 at home during the day-time hours. These assumptions give θ_1 = 40/112, θ_2 = 56/112, and θ_3 = 16/112.

The impact of intraday variation on pollution measures

Some intraday variation in the pollution data is captured by using the day-time and night-time means. However, there does not seem to be any theoretical reason for using the mean of the pollution distribution to measure the dose. In fact, a simple example illustrates that using the mean imposes a linear restriction on the dose-response function. Assume that the pollution exposure for some individual is variously POL1, POL2, and POL3 over the time period. Let the probability of each level occurring be f1, f2, and f3, respectively. Then, the mean equals f1*POL1 + f2*POL2 + f3*POL3. Next, let the dose-response function be linear in parameter space. Write it as

$$H = \delta_0 + \delta_1(f1*POL1) + \delta_2(f2*POL2) + \delta_3(f3*POL3)$$

for some individual in some time period. If $\delta_1 = \delta_2 = \delta_3$, then using the mean is equivalent to entering the probability distribution. On the other hand, different δ's would indicate that a mean model incorrectly restricts the health response to changes in average pollution; i.e., the response depends on how

the mean changed. The problem with this method is that we have no guidance on the number of δ's to specify. Still, this type of analysis may provide insights into the health response of distributions of air pollution.

IV. Empirical Results

The impacts intraday variability in Pollution exposures

In Table 2 we display the parameter estimates from several models. Only data from the first follow-up period was used to estimate the parameters. While the qualitative conclusions are similar for the second follow-up period, we could not statistically pool the data from the first two periods. The estimates based on the third and fourth period observations were quite different than those obtained from the first two. In particular, most of the parameter estimates were sensitive to the alternative specifications. This problem is apparently symptomatic of some type of survey bias, perhaps because respondents lost interest after the first two periods.

The specifications presented in Table 2 differ in the type of pollution measures entered. Several other independent variables could be selectively entered into the specifications. Those presented here are representative of the literature. The pollution coefficients are not particularly sensitive to any of the measures, except the seasonal dummies and, the weather variables. Selectively dropping these variables can change the sign of the pollution measures in some of the specifications. The weather variables and the seasonal dummies are statistically

significant in each specification, however.

The first specification represents a “traditional” air pollution dose-response function. The air pollution is assigned to the respondents home location code. The variable labels represent average ozone (OZMU) and average SO2 (SOMU), where the average is computed using data from the entire day. Fourteen of the eighteen coefficient estimates exhibit p-values of less than .05. As in other studies using a sulfur-oxide term, SOMU has a negative sign but is insignificant.

The remaining coefficient estimates are remarkably stable across different specifications. They show, all else equal, that:

- older respondents have fewer expected restricted activity days due to respiratory symptoms and disorders [E(NRRAD)],
- years of education do not affect E(NRRAD),
- the E(NRRAD) is lower for males,
- respondents in higher income classes have a lower E(NRRAD),
- respondents with good perceived health have a lower E(NRRAD),
- previous smokers have a greater (or insignificant) E(NRRAD),
- cooler temperatures and more rain increase E(NRRAD),
- when respondents are exposed to irritants at work, E(NRRAD) diminishes,
- respondents who exercise regularly, reduce E(NRRAD), and
- the greatest E(NRRAD) occurs during weeks 17-24, which is from the end of September to the middle of November.

One of the most interesting estimates is the coefficient on EXER. If individuals can reduce their expected number of respiratory related activity days by "expenditures" on regular exercise, this may give analysts an avenue for assessing the benefits of a cleaner environment.

Specifications (2), (3), (4), (5), and (6) illustrate the impacts of using different assignment methodologies (or weights) for the pollution terms. As indicated above, the actual dose is hypothesized to be some combination of the air pollution at home during the day (DOZLMU and DSOLMU), the air pollution at home during the night (NOZLMU and NSOLMU), and the air pollution at work during the day (WDOZLMU and WDSOLMU).

We used the mean of the natural logarithm of ozone and SO2 (all variable end with "LMU") because the log of the data appeared to be more normally distributed than the levels; thus, using the mean of the logs is a better measure of "the central tendency in the data."

The impacts of changing the θ's are dramatic on the estimated coefficients for ozone and SO2. In specification (2), $\theta_1 = \theta_3 = 0$ and $\theta_2 = 1$. The respondents are assigned the mean pollution at their homes computed over the day-time hours (11:00 am - 5:00 pm). The coefficient on DSOLMU remains insignificant, but becomes positive, and the likelihood function rises (the negative falls) slightly. Since the maximum reading usually occurred during this time interval, this specification is similar to [6], who used the average of daily maximums.

The third specification shows that, when the respondents are assigned a dose based on their home location pollution average over the night-time hours (5:00 am - 10:00 am and 6:00 pm - 12:00 pm), the ozone influence remains stable and the SO2 coefficient remains insignificant. The SO2 coefficient estimate jumps noticeably in magnitude, however, and the likelihood function continues to rise.

$\theta_1 = 1$ and $\theta_2 = \theta_3 =$ 0 in specification (4). The SO2 term is significant and of similar size to the one exhibited in (3). The coefficient estimate on the ozone term is also significant and about the same size as the estimate in (2) and (3).

Specifications (5) and (6) show the impact of non-zero **θ´s.** The **θ´s** are constructed a priori in specification (5), while in six they are estimated using a grid search. The log of the likelihood functions are the same up to the second decimal point. When comparing the coefficient estimates in (5) and (6) to the estimates obtained with the other models, we see that the impact of the pollution terms increases as the measures approach “real world” exposures.

The empirical significance of the alternative models is displayed in Table 3. Predictions of the expected value of NRRAD from each specification for one thousand identical individuals are shown. The predictions differ by the, type of change in ozone and SO2.

A change in just ozone (Predict2) can change the prediction on E(NRRAD) by from 1.928 per thousand to 10.671 per thousand; a difference of over 400%. Similarly, a change in just SO2 (Predict3) can change the E(NRRAD) per thousand from .356 (or -2.393, using specification 1) to 9.246; over 2000%. Clearly, the choice of the pollution measure can have a dramatic impact for policy analysts.

An analysis of non-mean models

As noted above, it is possible to test the mean specification. Based on the mean and standard deviation estimates of the logged data and assuming both pollutants were lognormally distributed, we computed the probability that the pollutants would fall into various categories. For ozone, we chose four categories; 0-5, 5.01-20, 20.01-60, and greater than 60. For SO2, we used 0-5, 5.01-10, 10.01-25, and greater than 25. The probabilities were computed for each of the distributions used above (i.e., day-time work, day-time home, and night-time home) and then averaged using the maximum likelihood estimates for the **θ´s.** The specifications with the probabilities entered as dependent variables did not significantly improve the model.[9] This was true for both ozone and SO2, indicating that the exposure time weighted mean model can not be rejected. Evidently, the distributional aspects of the air pollutants are adequately captured by specification (6) for the data analyzed here.

V. Conclusions

The primary conclusion of this paper is that predictions from alternative estimated dose-response functions differ substantially, depending on how pollution exposures are measured and assigned to individuals. The exposure varies because individuals" locations and air pollutants are not constant throughout a day. Dose-response specifications that use a weighted average of pollution experienced during the day at home, during the day at work, and during the night at home statistically outperform more traditional models. Moreover, the weighted average models indicate that the pollutants adversely affect human morbidity more than traditional models.

Our results indicate that sulfur-dioxide adversely affects human health. This finding is different from previous studies. The apparent reason for the difference is our treatment of the variable nature of the pollution. This particularly appealing, in the case of SO2, because SO2 is more localized than ozone. Hence, too much aggregation in the pollution data would mask the strength of the influence. By disaggregating the data, we have, hopefully, uncovered the true relationship.

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Endnotes

1. Portney and Mullahy find nonlinearities in the dose-response function. The marginal responses to increases in ozone are greater when equations are estimated with the data reflecting higher (greater than .05 pphm) ozone concentrations. They also find that the elasticity of the expected value of the respiratory RAD is not constant with respect to changing ozone as implied by the simple poisson distribution.

2. Survey respondents provided information on the number of visits to a doctor, the travel time to the doctor, visits to emergency rooms, and other "cost" measures. Gerking and Stanley [ ] use these data to estimate a willingness to pay for reduced air pollution expression that is based on an averting behavior model of consumer choice.

3. A statistical test indicated that the smokers could not be pooled with the non-smokers using a dummy variable to reflect smoking status.

4. An independent benchmark, the Regional Air Monitoring System (RAMS) data, was used to assess the quality of the SAROAD data. The RAMS data, which were collected in the late 1970s, but not during the time of the health survey, were subjected to extensive quality control and more accurately measure airborne pollutants. During the time that both systems were operational, the ozone readings between RAMS and nonRAMS data exhibit zero order Pearson correlations in the range of .3 to .6 for the hourly data. These correlations improve substantially as the data are aggregated to the daily and weekly level. Hence, there is every reason to expect that our use of the SAROAD data over a two-week period accurately measures the air pollution dose.

5. We did not model the weather as changing over the course of the day. It probably should be. However, the methodology proposed here facilitates a comparison to previous studies.

6. We computed Chi-square tests of distributional independence for all possible aggregations of the daily data by monitoring station. In the vast majority of cases, we cannot reject the hypothesis that the 11 am - 5 pm data come from the same distribution. Similarly, we cannot reject the hypothesis that the 5 am - 10 am and 6 pm - 12 pm data come from the same distribution. The hypothesis that all the daily .observations are generated by the same distribution was rejected in most cases, however.

7. The p-values are based on the variance-covariance matrix computed directly from the maximum likelihood estimates. They do <u>not</u> reflect a correction like in [6] or [3].

8. To identify the appropriate distribution of the data, we estimated the “transformed” mean and standard deviation and the transformation parameter. These parameters are based on the powernormal distribution, which utilizes the Box-Cox power transformation. The Box-Cox transformation facilitates a test between normal and lognormal distributions. In the majority of cases for SO2, the lognormal distributional assumption could not be rejected. With respect to ozone, we found that, usually, both the normal and lognormal distributions could be rejected. However, the transformation parameter was closer to O (indicating lognormal), than to 1.

9. We tested the probability model three ways. Firstly, by entering the probabilities for just ozone. Then, by entering the probabilities for just SO2, and, finally, by entering both. None of the Chi-squares, comparing twice the difference in the log likelihood values, indicated rejecting the linear constraint imposed by the mean specifications.

Table 1.
Variable Descriptions and Summary Statistics
Non-smokers, First Follow-up Period.
(Observations = 597)

Variable	Description	Mean	StDev.	Minimum	Maximum
AGE	Age in years	38.88	13.37	18	65
EDUC	Years of school	13.32	2.93	0	24
SEX	1 if male	.49	.50	0	1
WHITE	1 if white	.78	.43	0	1
INCOME	Income category	6.06	1.48	1	8
PHEALTH	1 if perceived health good	.93	.30	0	1
PSMOKE	1 if previous smoker	.18	.39	0	1
TEMP	Average temperature	60.40	20.84	13	83
RAIN	Average rainfall	.63	.57	0	2.15
IRR	1 if irritants at work	.34	.47	0	1
EXER	1 if exercise regularly	.11	.31	0	1
S1	1 if weeks 1 - 8	.26	.44	0	1
S2	1 if weeks 9 - 16	.30	.46	0	1
S3	1 if weeks 17 - 24	.13	.34	0	1
S4	1 if weeks 25 - 32	.17	.38	0	1

NRRAD Number of respiratory related restricted activity days during follow-up period two.

Frequency for NRRAD

Value of NRRAD	Frequency
0	558
1	39
2	9
3	7
4	6
5	2
6	3
7	4
8	1
9	0
10	0
11	1
12	0
13	0
14	0

Mean = .241

Table 2.
Alternative Coefficient Estimates of the Dose-Response Function
Dependent Variable = NRRAD

Variable	(1)	(2)	(3)	(4)	(5)	(6)
AGE	-.033*	-.032*	-.036*	-.030*	-.034*	-.033*
EDUC	-.018	-.024	-.015	-.024	-.021	-.022
SEX	-.083*	-.121	-.048	-.118	-.060	-.074
WHITE	.617*	.676*	.798*	.734*	.704*	.692*
INCOME	-.203*	-.199*	-.188*	-.164*	-.192*	-.191*
PHEALTH	1.146	1.266*	1.201*	1.326*	1.268*	1.284*
PSMOKE	.219*	.238	.181	.129	.185	.188
TEMP	-.054*	-.042*	-.038*	-.037*	-.040*	-.041*
RAIN	.853*	.957*	.716*	.956*	.982*	1.031*
IRR	-.302*	-.319*	-.214	-.273	-.229	-.245
EXER	-.888*	-.863*	-.800*	-.819*	-.854*	-.861*
S1	-2.441*	-2.681*	-2.321*	-2.536*	-2.867*	-2.941*
S2	1.318*	.773	1.421*	.796*	.853*	.719*
S3	1.565*	1.268*	1.575*	1.107*	1.516*	1.446*
S4	.841*	1.036*	1.057*	1.086*	1.410*	1.424*
OZMU	.037*					
SOMU	-.028					
DOZLMU		.951*				
DSOLMU		.065				
NOZLMU			.989*			
NSOLMU			.407			
WDOZLMU				.905*		
WDSOLMU				.367*		
OZLAVE1					1.692*	
SOLAVE1					.587*	
OZLAVE2						1.708*
SOLAVE2						.571*
CONST	1.043	-2.442*	-2.858*	-3.568*	-5.560*	-5.705*
θ_1	na	0	0	1	40/112	.4
θ_2	na	1	0	0	56/112	.4
θ_3	na	0	1	0	16/112	.2
-L like	270	269.5	268.8	269.4	264.5	264.5

*Indicates that the p-value is less than .05.

Table 3.
Predicted Reductions in the Expected Value of NRRAD
Per 1000 People by Specification

	(1)	(2)	(3)	(4)	(5)	(6)
Predict1	.637	2.197	14.294	5.418	16.351	12.940
Predict2	2.290	1.928	8.188	3.089	10.671	8.546
Predict3	-2.393	.356	8.119	3.022	9.246	7.186

Notes: The predictions are based on the following initial values: AGE=40, EDUC=12, SEX=1, WHITE=1, INCOME=6, PHEALIH=1, PSMOKE=0, TEMP=70, RAIN=.5, IRR=0, EXER=0, S1=1, S2=0, S3=0, and S4=0. The initial value for ozone is 40, while the initial value for SO2 is 20. The predictions are per 1000 people, where:

Predict1 is based on reducing ozone to 30 and SO2 to 10,
Predict2 is based on reducing ozone to 30 and maintaining SO2,
Predict3 is based on reducing SO2 to 10 and maintaining ozone.

Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Trade-offs*

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Abstract

This study develops a methodology for measuring the values that individuals place on morbidity risk reductions and applies it to the measurement of the benefits from reducing the risks of contracting chronic bronchitis. The survey methodology involves the use of an iterative computer program that presents respondents with a series of pairwise comparisons which are individually designed to measure respondents' marginal rates of substitution for chronic bronchitis risk reduction. The approach is innovative in that it measures the rates of trade-off for chronic bronchitis risk reduction in terms of the risk of an automobile accident fatality, as well as in dollars. Since it generates estimates for each individual, it can reveal distributions of benefit measures rather than simply a population mean estimate. The resulting rates of trade-off for chronic bronchitis and auto fatality risks suggests that the risk of a chronic bronchitis case is worth 32% of the comparable risk of death, as measured by the median trade-off rate. When risk reduction for chronic bronchitis is compared to a cost of living increase, the median rate of trade-off is $457,000, whereas the comparison between automobile fatality risk reductions and cost of living increases yielded a median rate of trade-off of $2.29 million. The results across different risk-risk and risk-dollar trade-offs were internally consistent.

1. Introduction

Over the past decade economists have devoted substantial attention to the implicit valuation of health outcomes. These analyses of risk-dollar trade-offs have relied in large part on market-based data.[1] For example, wage-risk trade-offs have been used to analyze the implicit value of fatalities and the average nonfatal job accident risk. Similarly, economists have analyzed the trade-offs implied by seat-belt usage decisions to infer a value of life.[2]

Although studies using market data provide useful benchmarks for health risk valuation, they do not resolve the issue of how government agencies should attach benefit values to health outcomes for which we do not have good market data. This omission is particularly important for government agencies, such as the U.S. Environmental Protection Agency (EPA), which generally focus on policy contexts in which market forces are believed to not be fully effective. For these situations, no useful market trade-off data may be available. Nevertheless, economic analysts would like to select the efficient project mix, and some benefit measure is required to perform such an analysis. In recent years, a large number of studies have addressed these benefit issues using non-market techniques, thus greatly expanding the range of benefit components that can be valued.[3]

This study makes several contributions to the literature on non-market techniques for benefit valuation. First, we develop a methodology for measuring the benefits of reducing the risks from various types of morbidity effects. The methodology uses an

iterative computer program to ascertain the points of indifference for consumers who are asked to trade off the reduced morbidity risk with increases in other attributes of a location decisions, such as an area's cost of living and the risk of an automobile fatality.[4]

Second, we apply the methodology to an important health benefit valuation problem, that of estimating the value of reductions in the risk from chronic bronchitis, one central type of chronic obstructive pulmonary disease alleged to be a major adverse effect of ozone pollution exposure. Most previous studies of health valuation focus on acute health effects, such as accidental death, rather than chronic diseases whose effects are more difficult to communicate to potential victims.[5]

Third, our approach yields the entire distribution of consumer values for chronic bronchitis risk reduction, rather than just the mean valuations which can be derived from market-based approaches to the problem. This information is important for policy makers in situations where consumers place. widely divergent values on reducing risk.

Fourth, because chronic disease effects are difficult to communicate to potential sufferers, it is important to use a methodology that adapts to whether subjects understand the valuation task being asked of them. By administering the questionnaires interactively on a computer, our approach allows us to build in several tests of task comprehension that, if failed, provide additional information before proceeding with the questionnaire.

Finally, our methodology produces values for morbidity risk reduction in terms of trade-offs with several other metrics besides money. In our chronic bronchitis application, we measure trade-offs with the risk of automobile fatalities, as well as with a dollar measure derived from changes in the cost of living. Many policy-makers are hesitant to base decisions on benefits denominated in dollars, and they may be more willing to implicitly consider benefit values when measured in units of a common risk such as death. Converting all health outcomes into death risk equivalents facilitates cost-effectiveness analysis by calculating the cost per statistical life equivalent saved, and it addresses concerns with respect to dollar pricing. Even if the morbidity valuations are elicited in terms of trade-offs between risks, they can still be converted into dollar values by using hedonic measures of the value of the comparison risk if that comparison risk is death (with the appropriate application of sensitivity analysis to the assumed values of life used to make the translation).

There are reasons to suspect that consumers may have fewer difficulties with the task of specifying rates of trade-off of one risk with another, as opposed to trading off a risk with a certain dollar amount. The risk-dollar trade-off task sometimes produces alarmist responses from subjects who cannot envision that they would voluntarily subject themselves to a higher risk of a serious morbidity effect for a finite amount of additional income.[6] Dollar valuation tasks also are difficult to design in ways that subjects will find analogous to real choice situations,

and they may offer biased responses to questions that do not force them to pay for the risk reduction being valued. There is a final reason to prefer the risk-risk trade-off approach. To the extent that consumers are equally adverse to the risks from different types of risks, asking them to trade off one risk against another produces rates of trade-off which measure the relative value to them of the two risks without regard to the risk aversion which enters in trading off uncertain health risk with certain dollars. In this sense the risk-risk trade-offs provide values which are not as heavily influenced by the consumers' attitudes towards facing risks per se.

The outline of this paper is as follows. Section 2 provides an overview of the study design and the sample. Section 3 describes the risk-risk trade-offs whereby respondents put their chronic morbidity valuations into auto death equivalents. In Section 4 we describe the direct estimates of risk-dollar trade-offs for chronic bronchitis obtained by asking respondents to trade off chronic bronchitis risks with either the area's cost of living or property damage from storms. As a check of the validity of the approach, we provide evidence on auto fatality risk-dollar trade-offs in Section 5. These implicit value of life numbers are tested against those in the literature to assess the valibity of the survey approach. In Section 5 we also convert all of our results for the value of chronic bronchitis to dollar equivalents. Section 6 concludes the paper.

2. Study Design and Sample Description

General Approach

We used a sample of 593 shoppers from a blue-collar mall in Greensboro, North Carolina to measure willingness-to-pay values for reducing the probability of contracting chronic bronchitis. The subjects made four series of pairwise comparisons of different locations where they could live with the locations differing in two attributes. In most of these comparisons, one of the locational attributes varied was the probability of contracting chronic bronchitis.

The first series of questions yielded a rate of trade-off between decreases in the risk of chronic bronchitis (CB) and increases in the risk of an automobile fatality, thus providing what we call a "risk-risk" trade-off. The second series of questions determined a "risk-dollar" trade-off, where the reduction in the risk of CB was achieved at the expense of a location with a higher cost of living.

If subjects were found to more easily trade off a reduced CB risk with a higher auto fatality risk than with a cost of living increase, we wanted to sort out whether this result was due to the fact that the cost-of-living differences were measured in dollars or that they were given with certainty (that is, with no risk involved over dollar gambles). Thus, our third series of questions asked subjects to trade off reductions in the CB risk with increases in a lottery on dollar losses expressed as a risk of storm damage, where if a storm were to occur, it would cause $2,000 of damage to the subject's home and belongings. Finally,

in order to compare the CB risk--auto fatality risk trade-offs with the risk-dollar trade-offs, it was useful to obtain a dollar measure of the value of reducing the risk of automobile fatalities. This fourth series of questions provided a rate of trade-off of risk reduction in automobile fatalities to increases in a location's cost of living.

The results from these four series of questions allows us to address the following questions:

* What is the distribution of CB risk--death risk trade-offs?
* What is the distribution of CB risk--(certain) dollar trade-offs?
* What is the distribution of CB risk--(uncertain) dollar trade-offs?
* Which of these three trade-offs is easier to elicit accurately from consumers?
* What is the distribution of death risk--(certain) dollar trade-offs?
* How does the distribution of CB risk--(certain) dollar trade-offs compare with the distribution of CB risk--dollar trade-offs derived from combining the CB risk--death risk trade-offs with the death risk--(certain) dollar trade-offs?
* How does the distribution of CB risk--(certain) dollar trade-offs compare with the distribution of CB risk--dollar trade-offs derived from combining the CB risk--death risk trade-offs with the values of life derived from wage hedonic studies?

It should be noted that the first question is the most important one to answer because it addresses the use of an

alternative metric to dollars for measuring morbidity risk willingness-to-pay values, that of another health risk, namely death. For cost-effectiveness purposes, it is not necessary to go beyond the death risk metric, as alternative policy initiatives can be compared on the basis of this metric rather than dollars. However, if the CB risk values measured in death risk units translate closely to the direct dollar valuations of reducing CB risks that we obtain, policy makers can be more confident in the reasonableness of the risk-risk valuations.

In order to understand our empirical results that allow responses to the questions above, it is first necessary to carefully describe the design of the survey and sample.

Methodology

The task of eliciting individuals' valuation of chronic bronchitis is not straightforward. The first problem is that few individuals fully understand the health effects of chronic bronchitis. Second, once given this information, they may not have sufficient experience in dealing directly with such trade-offs to give meaningful valuation responses. To accommodate these difficulties, we developed an interactive computer program that would inform consumers as well as elicit trade-off information.

Three different questionnaires were used, but for concreteness let us focus on what we will designate Questionnaire A. After acquainting the respondent with the computer, the program elicits information regarding the respondent's personal characteristics (e.g., age) . A substantial portion of the

questionnaire (about 40 questions) is then devoted to acquainting the respondent with the health implications of chronic bronchitis and the nature of the trade-offs that would be encountered. These questions elicit the respondent's familiarity with chronic bronchitis, information on smoking history, and provide a detailed summary of the health implications of chronic bronchitis.

The thirteen principal health implications of chronic bronchitis are summarized in Table 1. The chronic bronchitis disease classification includes a variety of illnesses of differing severity. Our intent was not to value each possible combination of systems, but rather to establish a methodology that could be used to value this and other adverse health effects. Consequently, our valuation procedure pertains to the set of symptoms summarized in Table 1, but the broader purpose of our analysis is to develop a methodological approach that is more generally applicable to other patterns of chronic bronchitis, as well as to different diseases such as cancer.

Since chronic bronchitis takes many forms, this study focused on the most severe chronic morbidity effects.[7] Thus, the survey's focus is on the adverse health outcomes at the extreme and of the cluster of diseases within the chronic bronchitis grouping. Because a quick overview of these effects may not be fully comprehended by respondents, in each case subsequent questions ascertain the respondents' assessed disutility ranking of each outcome in a linear 49-point scale. The purpose of these questions is not to establish attribute-based utilities, but to

Table 1

Health Implications of Chronic Bronchitis

1. Living with an uncomfortable shortness of breath for the rest of your life.
2. Being easily winded from climbing stairs.
3. Coughing and wheezing regularly.
4. Suffering more frequent deep chest infections and pneumonia.
5. Having to limit your recreational activities to activities such as golf, cards, and reading.
6. Experiencing periods of depression.
7. Being unable to do the active, physical parts of your job.
8. Being limited to a restricted diet.
9. Having to visit your doctor regularly and to take several medications.
10. Having to have your back mildly pounded to help remove fluids built up in your lungs.
11. Having to be periodically hospitalized.
12. Having to quit smoking.
13. Having to wear a small, portable oxygen tank.

encourage respondents to think carefully about the health implications of chronic bronchitis and their own view of the effect of this disease on their well-being.

At this point in the questionnaire, the respondents confront the first of two set of trade-off questions. Individuals are presented with a choice of moving to one of two alternative locations which differ in terms of their chronic bronchitis risk and automobile accident risk. To ensure that respondents would be willing to consider making such a move at all, they were told that these two locales posed a lower risk of both outcomes than their current place of residence.

Since risk levels differ across individuals, the program elicits information regarding individual activities that are likely to influence their person-specific risks, such as smoking habits (for chronic bronchitis) and mileage driven per year (for auto accident deaths). The program then informs the respondents that the probabilities presented in subsequent questions are calculated based on <u>their</u> responses to the earlier risk-related activity questions, even though the same risks are actually presented to all **subjects.**[8] This procedure increases the extent to which the stated risk levels are taken at face value, while facilitating the comparison of risk trade-offs across subjects because they all responded to the same risks.

To ensure that respondents understand the task before proceeding to questions in which one location is lower in one risk but higher in the other risk, they are first presented with a dominant choice situation. Let the notation (x,y) denote a

locale where the chronic bronchitis probability is x/100,000 and the automobile death risk is y/100,000. The actual survey did not present the choices in such abstract terms, but this notation makes the exposition of the survey structure simpler.[9]

To ascertain whether respondents understand the task, they are first asked whether they prefer Area A with risks per 100,000 population of (75, 15) or Area B with risks (55, 11). Since each of the Area B risks is lower, this alternative is dominant. Respondents who do not comprehend the task and incorrectly answer that they prefer Area A are sent through a series of questions that explain the structure of the choice in more detail.

The performance with respect to the dominance question was quite good. Over four-fifths of the sample gave a correct response to the dominance questions on their initial attempt. After being given additional information, fewer than one percent of them gave an incorrect answer, and these respondents were excluded from the sample since they did not understand the interview task.

The program then proceeds with a series of pairwise comparisons in which the attributes are altered based on the previous responses until indifference is achieved. The computer program used tabular summaries, but for expositional purposes we will consider the abstract formulation of the trade-offs.

A Model of State-Dependent Utilities

Consider the following model of state-dependent utilities. Let subscript a denote Area A and b denote Area B. Also, let U(CB) be the utility of a case of chronic bronchitis, U(D) equal

the utility of an auto accident death, and U(H) equal the utility of being healthy (i.e., having neither CB nor an auto accident). To simplify this exposition, we assume that contracting CB and dying from an automobile accident are mutually exclusive events. Also, let X_a denote the probability x/100,000 for Area A and Y_a denote the probability y/100,000 for Area A, and let x_b and y_b be defined similarly. The survey continually modifies the choice pairs until subjects reached the situation where

(1) $$X_aU(CB) + Y_aU(D) + (1 - X_a - Y_a)U(H) = X_bU(CB) + Y_bU(D) + (1 - X_b - Y_b)U(H).$$

Our general objective is to establish the death risk equivalent of chronic bronchitis. If we assume for concreteness that $X_a > X_b$ and $Y_b > Y_a$ (no loss of generality), then

(2) $$(X_a - X_b)U(CB) = (Y_b - Y_a)U(D) + (X_a - X_b + Y_a - Y_b)U(H),$$

or

(3) $$U(CB) = \frac{Y_b-Y_a}{X_a-X_b} U(D) + (1 - \frac{Y_b-Y_a}{X_a-X_b})U(H) \quad .$$

If we define the rate of trade-off between CD and D as t_1, SO that

(4) $$t_1 = \frac{Y_b-Y_a}{X_a-X_b} \ ,$$

we obtain the result that

(5) $$U(CB) = t_1U(D) + (1 - t_1)U(H) \ .$$

The utility of CB cases has been transformed into an equivalent lottery on life with good health and death, for which we have a well-developed literature.

Survey Structure

Now consider the first set of paired comparison questions presented in Questionnaire A after the dominant choice question described above. In this case, respondents are given the choice between Area A with risks (75, 15) and Area B with risks (55, 19). Suppose that Area B is preferred in this example. Area B has the lower chronic bronchitis risk and higher auto accident risk; therefore, in subsequent questions the program raises the CB risk in the preferred Area B until indifference is achieved. If in the original choice the subject prefers Area A, in subsequent questions the program lowers the auto death risk in Area B until the point of indifference is reached.

Suppose that after considering a series of such comparisons the subject reaches indifference where he views the risk (75, 15) as being equivalent to (65, 19). Using equations 4 and 5 above, this would imply that

$$t_1 = \frac{19-15}{75-65} = 0.4$$

and

$$U(CB) = 0.4U(D) + 0.6U(H).$$

The second set of paired comparison questions in Questionnaire A focuses on the more traditional risk-dollar trade-off involving CB and cost of living. Area A has the same

cost of living as the respondent's present residence, but Area B has a cost of living that is $80 higher, yet poses a lower CB risk X_b. If in the initial question Area B is preferred, Area B's CB risk is increased until indifference is achieved. Similarly, if Area A is preferred, Area B's cost of living is reduced until reaching the point of indifference.

In the context of a state-dependent utility function with two arguments, health status and income, we have

$$X_aU(CB) + (1 - X_a)U(H) = X_bU(CB,-\$80) + (1-X_b)U(H,-\$80).$$

If utility functions are additively separable in money and health, then

$$X_aU(CB) + (1 - X_a)U(H) = X_bU(CB) + (1-X_b)U(H) + U(-\$80),$$

which simplifies to

$$(X_a - X_b)U(CB) = U(-80) + (X_a - X_b)U(H),$$

or

$$U(CB) = \frac{U(-\$80)}{(X_a-X_b)} + U(H).$$

If we assume that utility is linear in money (with a coefficient equal to one) in establishing our health valuation scale, then we have

$$U(CB) = -L + U(H),$$

i.e., CB is equivalent to being healthy and suffering a financial loss tantamount to L dollars, where

$$L = \frac{-\$80}{X_a - X_b} .$$

This procedure to establish a risk-dollar trade-off rate involves two assumptions regarding the structure of utility functions. First, we assume additive separability with respect to money and health. Second, we assume that the dollar magnitudes treated are sufficiently small that utility is approximately linear in money. Since even risk-averse utility functions meet this test for small monetary changes,[10] we selected our health-risk levels so that the dollar magnitudes involved be small.

The structure of Questionnaire B is similar to Questionnaire A except the certain $80 loss in terms of living costs has been replaced by a lottery on $2000 storm damage loss. In this case, respondents must specify the storm damage probability that establishes an equivalent CB-storm damage pair. If we assume that respondents are risk-neutral, then the storm damage loss can be replaced by its expected value. The possible advantage over the cost-of-living approach is that respondents may be able to make more meaningful comparisons of two different lotteries rather than having one attribute -- the dollar payoff -- being non-stochastic. As with the first set of questions in Questionnaire A, if the consumer prefers Area B in the initial question, the program leads the consumer to indifference by increasing the CB risk of Area B until indifference is achieved. Similarly, when the consumer initially prefers Area A, the

program reduces the storm damage risk in Area B until reaching the point of indifference.

Questionnaire C repeats the first part of Questionnaire A, and these samples are pooled in the analysis below. The second set of questions addresses the more traditional death risk--dollar trade-off using auto deaths and cost-of-living trade-offs. The structure is similar to that of the second set of questions in Questionnaire A except that CB has been replaced by auto fatality risks so that respondents must reach the point that

$$U(D) = -L + U(H),$$

where

$$L + \frac{-80}{X_a - X_b}$$

as before. This portion of the study provides a direct comparability test with the literature on market-based values of life. The fatality risk--dollar trade-offs will also be used in conjunction with the chronic bronchitis--fatality risk trade-offs to establish a chronic bronchitis--dollar trade-off rate.

Table 2 summarizes the structure of the 3 questionnaires described above.

Sample Description

The interviews of the subjects were all done through an interactive computer program, thus avoiding problems of interviewer bias and promoting honest revelation of preferences. Response rates to sensitive questions, such as income level, were

Table 2

Summary of Survey Structure

Questionnaire A

Trade-Off	Units of Measurement	Procedure
1. Chronic bronchitis - auto deaths	Auto deaths per chronic bronchitis case	In the area with the higher auto accident risk, increase the bronchitis risk (to make that area less desirable) or reduce the auto accident risk (to make that area more desirable) until reaching in-difference.
2. Chronic bronchitis - cost of living	Dollar value per 1/100,000 reduced risk of bronchitis	In the area with lower bronchitis risk, increase the bronchitis risk (to make that area less desirable) or decrease the cost of living (to make that area more desirable) until reaching in-difference.

Questionnaire B

1. Chronic bronchitis - storm damage	Reduced probability of $2000 storm damage that is equivalent to one bronchitis case prevented	In the area with the higher storm damage risk, increase the bronchitis risk (to make that area less desirable) or reduce the storm damage risk (to make that area more desirable) until reaching indifference.

Table 2 (cont'd)

Summary of Survey Structure

Questionnaire C

1.	Chronic bronchitis - auto deaths (Same as Questionnaire A - Part 1)	Auto deaths per chronic bronchitis case	In the area with the higher auto accident risk, increase the bronchitis risk (to make that area less desirable) or reduce the auto accident risk (to make that area more desirable) until reaching indifference.
2.	Auto accidents - cost of living	Dollar value per 1/100,000 reduced risk of an auto accident	In the area with lower auto accident risk, (to make that area less desirable) or decrease the cost of living (to make that are more desirable) until reaching indifference.

much higher than those usually achieved with face-to-face interviews. In addition, subjects were not concerned with whether their responses impressed the interviewer. Use of a computer also made it possible to ask a sequence of questions to ascertain the appropriate marginal rates of substitution.

The sample was recruited for the study by a professional marketing firm at a mall intercept in Greensboro, North Carolina. This locale has a representative household mix and is used as a test marketing site for many national consumer brands. This firm and locale have been used successfully in two previous studies by the authors.[11] Use of such a consumer sample also yields more reliable responses to issues such as the valuation of property damage from storms than would a student sample or a sample from a city with an unrepresentative population, such as the college-oriented cities of Evanston, Illinois, or Chapel Hill, North Carolina.

Table 3 provides a glossary of the variables and the associated sample statistics. Questionnaires A and C had a similar mix of respondents, with a mean age in the low thirties. a even split between males and females, two years of college education, a 50 percent married rate, about 0.6 children under 8 years old, a household size of 2.7 - 2.8, and a household income in the mid-range of thrifty to forty thousand dollars. Questionnaire B has a somewhat different mix because of the difference in the times at which the samples were recruited (e.g., week-end shoppers differ from day-time weekday shoppers). The Questionnaire B sample is about 10 years older, more likely

Table 3

Summary of Sample Characteristics

	Mean and Std. Deviations		
	Questionnaire		
Demographic Variables	A	B	C
AGE, in year	33.74 (12.42)	43.47 (12.68)	33.07 (11.66)
MALE, sex dummy variable	0.50	0.42	0.51
EDUCATION, years of schooling	14.02 (2.23)	14.32 (2.47)	13.79 (2.66)
MARRIED, married dummy variable	0.49 (0.50)	0.79 (0.41)	0.49 (0.50)
KIDS, number of children under 8	0.56 (1.00)	0.83 (1.04)	0.65 (1.07)
HOUSEHOLD, number of people in household	2.71 (1.25)	3.00 (1.16)	2.80 (1.23)
INCOME, annual household income in dollars	35,386.60 (19,009.95)	45,367.65 (20,335.54)	37,153.85 (21,333.80)
	194	204	195

to be married, and with a household income about $10,000 greater. As the last row of Table 3 indicates, each of the three samples had about 200 respondents, with combined sample for the study of 593.

3. Risk-Risk Trade-Offs

Table 4 displays the means and standard deviations of the trade-off rates implied by the indifference points of the subject responses. To go beyond these summary statistics, consider first set of trade-offs between CB and auto accident deaths. For this analysis Questionnaires A-1 and C-1 are pooled since the questions are identical.

Establishing a death risk metric for CB enables respondents to think in risk terms, avoiding the comparability problems that might be encountered if monetary attributes were introduced. Similarly, for policy purposes EPA can establish a death risk equivalent and establish cost-effectiveness ratios in terms of the cost per statistical death prevented. As indicated in Viscusi (1986), this cost-effectiveness index will provide a comprehensive measure of the policy impact and also avoid the political sensitivities of placing dollar values on all health outcomes. Once a uniform health metric is established, one can then compare the cost per life equivalent saved with various value-of-life reference points and decide whether the policy should be pursued if one wishes to take a benefit-cost approach.

Unlike market-based studies of the value of life, the survey technique yields information on the entire distribution of the valuations. Table 5 reports the deciles of the distribution for

Table 4

Rates of Trade-off Implied by Indifference Points

	Means and Std. Deviations		
Part B	Questionnaire		
Trade-off Rates	A	B	C
CB-Auto (A-1 & C-1), auto deaths per CB case	0.68 (0.82)	-	0.70 (0.95)
CB-Cost of Living (A-2), dollar value per 1/100,000 CB risk	8.83 (12.50)	-	-
CB-Storm Damage (B-1), number of $2,000 storms equal to one CB case	-	852.60 (1064.20)	-
Auto-Cost of Living (C-2), dollar value per 1/100,000 reduced auto accident risk	-	-	81.84 (168.54)
Sample Size	194	204	195

Table 5

Distribution of Chronic Bronchitis --

Auto Death Trade-Offs

Decile	Auto Death Equivalents per Chronic Bronchitis Case
.10	0.12
.20	0.20
.30	0.23
.40	0.27
.50 (median)	0.32
.60	0.40
.70	0.80
.80	1.00
.90	1.33
1.00	4.00
Mean	0.68
(St. error of mean)	(0.06)

respondents who gave consistent answers that converged to a particular trade-off value. Subjects whose responses indicated that they did not fully comprehend the valuation task were excluded from our sample.

Specifically, we excluded subjects who failed one of the following consistency checks:

1) they started the series of paired comparison questions by preferring one area, say Area A, and as Area B was made more desirable in subsequent comparisons they continued to prefer Area A, even on the last question of the series in which Area B dominated Area A on both attributes;
2) like inconsistency #1, they continued to prefer Area A in each comparison until the last one in which Area B dominated Area A in both attributes, yet on this last question they indicated indifference between Area A and Area B;
3) they indicated preference for one area, say Area A, on the first and all subsequent questions in the series (including the last one in which Area B dominated Area A), then when confronted with this inconsistency and asked to repeat the series of questions chose Area B in the first question (despite have selected Area A the first time they were given this question);
4) they indicated preference for one area, say Area A, on all questions in the series except the last one in the series (in which Area B dominated Area A) but including the next-to-last question (for which Area B easily dominated Area A on one attribute and Area A just barely dominated Area B on the

other attribute) , thus making it impossible to interpolate between the trade-offs implied by the last two questions to obtain an indifference point (because the last question yields no rate of trade-off); or

5) they expressed indifference between all pairs of areas in the series of questions, despite wide variation in their attributes.

Individuals who failed one of these inconsistency checks either did not understand the choice task, were not responding honestly, attached no value to one of the two attributes, or have non-monotonic preferences for one of the attributes. We assume that neither of the last two preferences attributes are possessed by any subjects, thus implying that answers which fail any of the five inconsistency checks do not represent the subjects' true preferences.

The requirement that the response pattern to the series of paired comparisons be internally consistent will lead to more meaningful estimates than if no such checks were imposed. About two-thirds of the sample converged to an indifference situation and had consistent responses, where this percentage was similar across all questionnaires.[11] These consistency checks distinguish our approach from the usual contingent valuation method in which respondents' answers are taken at face value without such formal tests of whether the subjects understood the valuation task and displayed consistent choices.

In evaluating the distribution in Table 5, first consider the respondent at the tenth percentile. This person viewed a

chronic bronchitis probability as being just as severe as a risk of an auto accident that was 0.12 as great. Thus, this individual would view a chronic bronchitis risk of 100/100,000 risk of 100/100,000 per year as being equivalent to the annual chance of being involved in an auto accident of 12/100,000.

Now examine the respondent at the other end of the distribution. This individual views a chronic bronchitis risk as being four times as severe as a risk of death, so that a 100/100,000 risk of CB would be viewed as comparable to a 400/100,000 risk of death. He or she gave consistent responses to the questions, but opted for the choice reflecting the highest CB valuation.

Many studies in the survey valuation literature exclude the tails of the distribution since they are tainted by extreme respondents such as this. Rather than discard such information altogether, we report the entire distribution, recognizing that the top and bottom deciles may be affected by a lack of complete understanding of the interview task. The reported distributions enable readers to assess how important outliers are within the context of the study and by focusing primarily on the median responses rather than the mean we avoid the distortion of our results by these outliers.

The response pattern in which CB was more highly valued than auto death risks was exhibited by the top two deciles for each questionnaire's response distribution. Such a pattern is not necessarily implausible. In addition to possibly misunderstanding the interview task, two explanations can be

offered. First, individuals might legitimately believe that such a severe chronic illness is a worse outcome than death. The health outcome described in Table 1 is quite serious and will have substantial duration. Their normal activities would be curtailed, medical interventions including hospitalization and possible reliance on a portable oxygen tank would accompany severe cases of CB, other illnesses would be more likely, and they would experience periods of depression.

The second possible explanation is that the respondents were establishing equivalences between different average risks in an area rather than different risks to themselves. The CB risk was characterized as an involuntary risk not under their control except for smoking, whereas the auto accident risk differs depending on one's driving habits and skills. Other studies suggest that individuals may have overly optimistic assessments of risks influenced by their actions, such as auto death risks, as discussed in Viscusi and Magat (1987). If this were the case, the perceived person-specific risk would be below the stated risk, causing an upward bias in the results in Table 5.

The median CB valuation is equivalent to 0.32 auto deaths. Because of the skewed nature of the responses, the mean value of 0.68 is more than double the median response. Regression analysis of the CB-auto death trade-off rates indicate no significant variation across subjects with respect to either demographic factors such as age, income, and education, or personal characteristics such as smoking habits. This result is neither surprising nor disturbing. Most individual attributes,

such as household income, should affect the CB valuation and the value of life similarly, and thus be unrelated to variation in the CB--auto death trade-off rates across subjects. Because there are no systematic differences among individuals in their risk-risk trade-offs, we can aggregate them into meaningful summary measures such as medians and means without the risk of drawing misleading conclusions from an unrepresentative sample.

The general implications of these results is a follows. Most, but not all, people regard the risk of chronic bronchitis as a less severe outcome than the risk of death. However, the prospect of a sustained chronic illness is viewed as a very severe outcome. Based on the median responses, the death risk equivalent of CB is. 0.32, and based on the mean response it is 0.68. The general order of magnitude of both the median and the mean is the same and is just below that of fatalities. As will be indicated in Section 5, these statistics can be transformed into dollar valuation equivalents using established value-of-life statistics.

4. Risk-Dollar Valuations of Chronic Bronchitis

The second approach that we employed to value chronic bronchitis was to establish risk-dollar trade-offs. The two approaches used were to establish the chronic bronchitis risk equivalent of a higher cost of living and to determine the relationship between chronic bronchitis risks and storm damage risks.

Consider first the cost-of-living results in Table 6. The first column of Table 6 lists the decile of the distribution.

Table 6

Distribution of Chronic Bronchitis -

Cost of Living Trade-Offs

Decile	Trade-Off Levels: Dollar Value per 1/100,000 Reduced Risk of Chronic Bronchitis (A-2)	Trade-Off Levels: Implicit Dollar Value per Case of Chronic Bronchitis
.10	1.50	$150,000
.20	3.00	$300,000
.30	3.50	$350,000
.40	4.00	$400,000
.50 (median)	4.57	$457,000
.60	5.33	$533,000
.70	6.40	$640,000
.80	8.00	$800,000
.90	20.00	$2,000,000
1.0	80.00	$8,000,000
Mean	8.83	$883,000
(St. error of mean)	(1.14)	($114,000)

Column two presents the increased dollar value in the annual cost of living that the respondent was willing to incur per 1/100,000 reduction in the annual probability of chronic bronchitis. If we multiply the results in column 2 by 100,000, we obtain the implicit dollar value per statistical case of chronic bronchitis.

As in the case of the risk-risk results, the response pattern is skewed so that the upper tail of the responses generates a mean valuation estimate in excess of the median. The results here indicate the average dollar value of chronic bronchitis is $883,000, with an associated standard error of $114,000. The $457,000 median of the distribution is just over half of the mean. Each of these values is below the usual estimates of the implicit value of life, which are reviewed in Viscusi (1986). These results follow the expected pattern, given the CB--auto death risk trade-off results reported above.

As in the case of the risk-risk trade-offs, the upper bound of the chronic bronchitis valuation estimates exceeds most estimates of the value of a fatality, as $8 million exceeds some but not all estimates of the value of life. More precise comparisons of all of the results using a dollar metric will be undertaken in Section 5.

The second set of CB risk-dollar trade-offs, which is reported in Table 7, uses storm damage risks as the dollar counterpart so that respondents must compare monetary lotteries and health status lotteries rather than certain monetary (cost of living) differences with health status lotteries. The first column of results gives the value of y for which a storm causing

Table 7

Distribution of Chronic Bronchitis --

Storm Damage Trade-offs

Decile	Equivalent $2000 Damage Probability (x100,000)	Implicit Dollar Value per Case of Chronic Bronchitis
.10	175.00	$350,000
.20	228.57	$457,140
.30	266.67	$533,340
.40	266.67	$533,340
.50 (median)	400.00	$800,000
.60	533.33	$1,066,660
.70	800.00	$1,600,000
.80	1,333.33	$2,666,660
.90	2,000.00	$4,000,000
1.0	4,000.00	$8,000,000
Mean	852.60	$1,705,200
(St. error of mean)	(91.93)	($183,860)

damage of $2000 with a probability of y/100,000 is equivalent to a chronic bronchitis probability of 1/100,000. A more meaningful metric is the expected storm damage that is equivalent to each CB case. This figure is obtained by multiplying the first column of results by the $2000 damage per storm damage event. The second column of results gives the dollar value per statistical case of chronic bronchitis, where these dollar values have been obtained using the storm damage costs.

A comparison of the distributions of implied CB valuations in Tables 6 and 7 suggests that the subjects may have found the storm damage lottery comparison to have been more difficult to make than the comparison with a non-probabilistic cost-of-living increase. The distribution derived from the storm damage lottery comparison stochastically dominates the distribution from the cost-of-living comparison, with both its median and mean almost double that of the cost-of-living distribution. Based on a comparison with the dollar values of avoiding automobile accident fatalities reported in next section, the CB avoidance values derived from the storm damage lottery questions appear to be somewhat high. Further, the standard error of the mean is about 50 percent higher for the distribution derived from the storm damage distribution than for the cost-of-lived based distribution of CB values. In any event, these results do not suggest that expressing dollar trade-offs in probabilistic form, as in the storm damage lottery, aids people in making risk-dollar trade-offs, which was our original hypothesis.

5. Trade-Offs Between Auto Deaths and Cost-of Living

A useful check on the survey methodology is to ascertain the implicit value of life using a direct fatality risk-dollar trade-off. This is done using automobile accident risks and cost of living in Questionnaire C-2, and the results of this exploration are reported in Table 8.

The median response of $2,286,000 is quite reasonable in view of the similar (in 1987 dollars) market-based estimate by Blomquist (1979), but the mean value of $8,184,000 seems rather large. The high mean estimate was generated by a portion of the sample with value of life estimates as high as $80,000,000. Such implausibly large estimates can occur because of the difficulty of the comparison task. Respondents are being asked to establish an equivalence between some annual chance of chronic bronchitis x/100,000 that is equivalent to an $80 cost-of-living increase. This is a difficult comparison to make. In contrast, the risk-risk questions focused on chronic bronchitis--auto accident risk comparisons of x/100,000 and y/100,000, where most respondents did not believe that the severity of outcomes differed by more than an order of magnitude.

The implicit dollar value of CB can be obtained by chaining the responses to questionnaire part C-1, which gives the CB-auto death trade-off, and part C-2, which gives the auto death--dollars trade-off. These results appear in Table 9. The median dollar value of each chronic bronchitis case is $800,000. The mean is much greater because there is one outlier with a $320 million value. This individual expressed extreme responses on

Table 8

Distribution of Auto Accident --

Cost of Living Trade-Offs

Decile	Dollar Value per 1/100,000 Reduced Risk of an Accident	Implicit Dollar Value of an Accident
.10	10.00	$1,000,000
.20	17.50	$1,750,000
.30	17.50	$1,750,000
.40	20.00	$2,000,000
.50 (median)	22.86	$2,286,000
.60	26.67	$2,667,000
.70	40.00	$4,000,000
.80	80.00	$8,000,000
.90	177.78	$17,778,000
1.0	800.00	$80,000,000
Mean	81.84	$8,184,000
(St. error of mean)	(14.40)	($1,440,000)

Table 9

Implicit Valuation of Chronic Bronchitis

Implied by CB--Auto Death and Auto Death --

Cost of Living Trade-offs

Fractiles	Questionnaire C Inferred CB Value
.10	$200,000
.20	$350,000
.30	$522,449
.40	$646,154
.50	$800,000
.60	$1,066,667
.70	$2,133,333
.80	$3,555,556
.90	$12,800,000
.99	$71,111,111
1.00	$320,000,000
Mean	$6,962,364
(Std. Error of Mean)	($2,977,373)
	(N = 112)

each component part, valuing each CB case at four times the amount of each death and having an implicit value of an auto fatality of $80 million. In each case, these were the highest values in the sample and the highest permitted by the Program, which indicates that this individual probably did not understand the valuation task.

As instructive summary of the results is provided in Table 10. For the results creating CB/auto death risk equivalents, the numbers have been transformed into implicit value-of-life terms using three different reference points: a $2 million value of life; a $3 million value of life; and a $5 million value of life. The $2 million figure is comparable to the median auto death risk valuation within the survey so that this estimate provides an internal comparison of the results. The $3 million figure is included since the recent estimates by Moore and Viscusi (1988) indicate that the labor market value of life is in the $2-$3 million range using BLS data, and this was the “best estimate” of the value of life in earlier work by Viscusi (1983). The $5 million reference point is the value of life figure obtained using new NIOSH data on job fatality risks, which Moore and Viscusi (1988) view to be superior to the BLS data.

The pattern displayed by the results is fairly similar. In each case mean valuations are at least double the value of the median. Although one would not expect symmetry in a distribution truncated at zero, the very high end responses observed appear to be due to response errors.

Table 10

Summary of Risk-Dollar Equivalents

	Direct Valuation Estimate	CB Estimate Using $2 Million Value of Life	CB Dollar Estimate Using $3 Million Value of Life	CB Dollar Estimate Using $5 Million Value of Life
CB/Auto Fatality:				
A-1 & C-1 (Median)	--	$640,000	$960,000	$1,600,000
A-1 & C-1 (Mean)	--	$1,360,000	$2,040,000	$3,400,000
CB/Cost of Living:				
A-2 (Median)	$457,000	--	--	--
A-2 (Mean)	$883,000	--	--	--
CB/Storm Damage:				
B-1 (Median)	$800,000	--	--	--
B-1 (Mean)	$1,705,200	--	--	--
CB/Dollars (Derived from CB/Auto Fatality and Auto/Cost of Living):				
C-1 & C-2	$800,000	--	--	--
C-1 & C-2	$6,962,364	--	--	--
Auto/Cost of Living:				
C-2 (Median)	$2,286,000	--	--	--
C-2 (Mean)	$8,184,000	--	--	--

The most clearcut divergence from plausible patterns is the mean value of life of $8,184,000 for the auto death\cost-of-living trade-off. Whereas the mean CB/auto values were roughly double the median, the mean auto/cost of living values were almost four times the size of the median, indicating a much more skewed distribution. As noted in the discussion of Table 8, this mean value was influenced in part by individuals with implied values of life as high as $80 million. These outliers suggest that for some People making meaningful trade-offs involving small cost-of-living differences and low risks of auto accident fatalities is a task they cannot handle effectively.

The valuation of chronic morbidity across the difference questionnaire approaches is quite similar for the case in which we use a $2 million value of life figure to transform the death equivalent statistics into meaningful dollar estimates. The median value for the CB/auto death risk trade-offs is $640,000, as compared with a median value of $457,000 for the CB/cost of living trade-off and a median value of $800,000 for the CB/storm damage results. These results are similar to the $800,000 median CB value that was obtained by chaining the CB/auto and auto/cost of living responses. Even with a higher value of life of $3 million, the CB/auto median of $960,000 is not out of line with the CB/cost of living and CB/storm damage results.

Once we move to the case where a $5 million value of life is used, the median dollar valuation of each CB case prevented is greatly increased to the $1,600,000. If EPA were to rely on, for example, the CB/cost of living results to value CB and then use a

value of life of $5 million without also using an appropriately adjusted CB value, this procedure could potentially understate the value of the CB cases prevented by a factor of three. By converting all outcomes to a health risk equivalence scale using a death risk metric, EPA avoids any distortion in the mix of targeted illnesses that might otherwise occur if the value of life number selected was incorrect.

6. Conclusion

Although market evidence remains our most reliable guideline for assessing the shape of individual preferences, such evidence is unavailable for many outcomes that are either not traded explicitly in markets or traded implicitly but in a market for which available data are not rich enough to identify the pertinent trade-off rates. Analysis of risk-risk and risk-dollar trade-offs using various types of simulated market choices provides a useful mechanism for establishing such values.

This study has developed a methodology for deriving morbidity valuation estimates based on the trade-off with another well-known risk, rather than forcing individuals to express trade-off rates between morbidity rate reductions and dollars, a task which is unfamiliar to most people. We presented several conceptual reasons why consumers should be able to more accurately convey risk-risk trade-offs than risk-dollar trade-offs, and the application of our methodology to the valuation of reductions in the risk of chronic bronchitis indicate that most individuals can make risk-risk trade-offs, even with a disease as

complicated and unfamiliar to healthy people as chronic bronchitis.

Although for the purpose of cost-effectiveness analysis there is no need to measure risk reduction value in terms of dollars, when we translated our risk-risk estimates into risk-dollar estimates using either survey results on auto accident risk reduction values or published value-of-life estimates, the distributions compared favorably, thus providing additional confidence in the reasonableness of the results derived from our methodology. While this study applied the approach to the valuation of only two risks, that of chronic bronchitis and an auto accident fatalities, the favorable results suggest that the methodology may be more widely applicable to other morbidity risks, such as various forms of cancer.

Footnotes

[1]See Viscusi (1986) for a review of the market trade-off literature.

[2]See analysis by Blomquist (1979) for an inventive use of seatbelt usage data to infer a value of life.

[3]Survey studies of various health and environmental risks include the seminal work by Acton (1973) as well as more recent studies often grouped under the designation "contingent valuation. " These recent analyses include: Brookshire, Thayer, Schulze, and d'Arge (1982) ; Cummings, Brookshire, and Schulze (1986); Fischhoff and Furby (1988); Gerking, de Haan, and Schulze (1988); Smith and Desvousges (1987); Viscusi and Magat (1987); Viscusi, Magat, and Forrest (1988); and Viscusi, Magat, and Huber (1987); and Fisher, Chestnut, and Violette (1989).

[4] In designing our survey, we used software from Sawtooth Software, Inc.

[5]For an important recent study of the valuation of health risks rather than mortality, see Berger et al. (1987).

[6]For example, see Viscusi, Magat and Huber (1987), pages 477-478.

[7]See Petty (1985) for a discussion of the distinction between chronic bronchitis, the related disease emphysema, and the broader disease category called chronic obstructive pulmonary disease. The authors selected the type of chronic bronchitis described in Table 1 after consulting closely with two lung specialists at Duke University Medical Center and visiting the Medical Center rehabilitation program for patients with severe lung diseases.

[8]At the end of the interview, subjects were carefully debriefed about this use of average rather than person-specific risks.

[9]Our past studies suggest that presenting the risk in terms. of the number of cases for a large base population is more comprehensible than giving risk levels such as 0.00075.

[10]See Arrow (1971).

[11]See Viscusi and Magat (1987) and Viscusi, Magat, and Huber (1987) .

[12]Probit analysis was used to identify personal characteristics that explain the division of subjects between those giving consistent and inconsistent responses. The only two significant variables in the equation are AGE and SMOKER, with older respondents less likely to give consistent responses and smokers more likely to respond consistently. These results may reflect the difficulty that older subjects have with the new interview technology (computers) and the greater thought that smokers have given to the implications of chronic bronchitis.

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